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SYMPOSIUM ON MASTOIDITIS.

THE SYMPTOMATOLOGY AND DIAGNOSIS OF ACUTE MASTOIDITIS.*

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Acute surgical mastoiditis is almost always secondary to otitis media, hence the symptoms are derived from the middle ear as well as the mastoid process.

Primary mastoiditis is such a rare disease that it will not be considered in this paper.

The symptoms of acute mastoiditis vary according to: *1.* age of the patient; *2.* type of the mastoid process; *3.* extent of the mastoid involvement; *4.* variety, type and virulence of the infection; and *5.* resistance of the patient.

The symptoms may be divided for the purpose of discussion into two groups: general and local.

The general symptoms are similar to those of other infections, *i. e.*, fever, chilly sensation, malaise, headache, insomnia, loss of appetite, restlessness, etc., and their severity is in direct proportion to the acuteness of the attack and the virulence of the infection.

The general symptoms are usually more marked in infants and young children in whom there may be added symptoms of cerebral irritation, such as crying out at night, marked irritability and even

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in some instances convulsions. These symptoms are explained by the close association of the middle ear and tympanic antrum with the cerebral structures through a patent petrosquamous suture, and the unstable nervous system of infancy and early childhood.

The general symptoms may be present in the early stages of the disease and then practically disappear when the patient gains control of the infection, or when the pathologic process becomes walled off.

The general symptoms are more marked in the virulent infections, particularly the exanthemata and influenza; and it is often difficult to differentiate between the symptoms due to the general infection and those caused by the local lesion.

In adults, the general symptoms are apt to be more marked where there is an involvement of a large absorptive area such as is found in highly pneumatic mastoid processes.

If the involvement of the mastoid process is gradual, the general symptoms may be slight. This is more apt to occur with the less virulent infections.

Adults suffering with some severe constitutional disease, such as syphilis, tuberculosis, cancer, nephritis and diabetes, may develop a low grade so-called latent mastoiditis secondary to middle ear disease with very little general disturbance; likewise, poorly nourished and dehydrated infants may develop middle ear and tympanic antrum disease without the usual general manifestations.

Ordinarily, an elevation of temperature is present in mastoiditis in infancy and early childhood, and usually it is remittent in type, and a peak of 104° or 105° is not uncommon in the early stage. On the contrary, an elevation of temperature is not usually found in mastoiditis of adults. Therefore, elevation of temperature is not particularly significant in infants and children, but it is a most important symptom in adults.

A continuance of elevation of temperature in either children or adults, however, otherwise unexplained, is usually indicative of mastoid involvement and possibly a complication.

A sudden elevation of temperature associated with a cessation of middle ear discharge usually indicates mastoid involvement.

Chilly sensations very often accompany elevations of temperature but distinct chills should be viewed with suspicion, as they usually indicate mastoid involvement plus a blood stream infection.

Fullness in the affected side of the head and headache are common symptoms in adults, and if severe are usually suggestive of mastoiditis.

Sleeplessness in adults is very significant, and very often is not due to pain. The patient awakens several times during the night, and in the morning looks tired and worn.

All of the general symptoms may be exaggerated in patients of low resistance.

The common local symptoms of acute mastoiditis are pain in and about the ear; tenderness over the mastoid process; purulent discharge from the ear; diminution of hearing; in some instances tinnitus; swelling of the soft parts about the ear, sometimes with displacement of the auricle; and canal and fundus changes.

The local symptoms of infants differ from those of adults owing to the nondevelopment of the mastoid process. It is therefore more correct to speak of an antritis than a mastoiditis. It is not until after the third year that the mastoid process begins to assume the adult type.

Pain and tenderness may be elicited in infants by palpation but it is difficult to differentiate between pain in the middle ear and pain in the tympanic antrum.

Swelling of the soft parts behind the ear is more common in infants and young children because of the spread of the inflammatory process through the patent mastosquamous suture.

The canal and fundus changes are not as characteristic in infants on account of the lack of development of the bony canal. The typical sagging therefore does not occur.

A low grade infection of the mastoid antrum may occur in infants and young children whose resistance has been lowered by some general disease, without local symptoms other than a persistent purulent discharge from the middle ear.

In adults, the local symptoms vary considerably according to the type of the mastoid process. The cellular mastoids, particularly the highly pneumatic, are more commonly involved by the acute suppurative inflammations.

The cellular structure may occupy almost the entire temporal bone and may even extend into neighboring bones. Access to these

cells is easily gained by the infecting micro-organisms, but drainage through the middle ear may be impossible in some cases, particularly from the distant cells.

This type of mastoid process is apt to give marked local symptoms. The discharge from the ear is profuse owing to the large area of lining membrane involved, and pain is very often severe, depending however upon the freedom of drainage. Owing to the thinness of the cortex, local tenderness is apt to be marked.

The symptoms may vary according to the extent of the mastoid involvement. The infection may pass from the mastoid antrum to an isolated area of the mastoid process and produce localized symptoms. This area or group of cells may become shut off from the main body of the mastoid through swelling of the mucosa lining the communicating channel. It is in these pent up areas that necrosis of bone is apt to occur with consequent exposure of underlying structures. Localized pain and tenderness are characteristic of this condition.

Swelling of the soft parts behind the ear with displacement of the auricle either forward or downward occasionally occurs due to an extension of the inflammatory process through the thin cortex. The swelling may be either edema or a subperiosteal collection of pus.

Swelling of the soft parts in front of the ear and beneath the temporal fascia may also occur by extension from the zygomatic cells in the highly pneumatic bones. This swelling is frequently accompanied by painful mastication and edema of the eyelids.

A painful swelling of the upper cervical region, just below the mastoid tip, may result from a rupture of a pneumatic cell or cells through the inner wall of the tip at the digastric groove, the so-called Bezold's mastoiditis. This is usually accompanied by a painful rigidity of the neck and a laterally flexed position of the head.

The non-cellular mastoid processes are not so apt to be associated with acute inflammation and when involved do not produce as many local symptoms as the pneumatic.

Pain is usually of a deep boring character. Tenderness is usually not present because of the thickness of the cortex. The middle ear discharge is less profuse and subperiosteal swellings are very infrequent.

In all cases, whether of the cellular or non-cellular type, the dis-

charge varies with the stage of the disease and the character of the infection. At first it is usually serosanguineous, then it becomes mucopurulent, and if the mucous membrane is eroded and the bone is involved, it becomes entirely purulent. As a general rule, the pneumococcic infections produce a more purulent discharge than the streptococcic.

The amount of the discharge is usually an indication of the extent of the inflammatory process and a persistent profuse discharge usually signifies a continuance or an extension of the infection, while a subsidence, particularly if associated with a diminution of pain, indicates a lessening of the infection.

A continuous discharge for two or three weeks, without other local symptoms, ordinarily indicates mastoid involvement.

A recurrence of discharge after a cessation is usually indicative of mastoid involvement, particularly if associated with pain or tenderness.

It is possible to have a mastoiditis without discharge from the ear. This may be explained by a sealing off of the middle ear from the antrum and mastoid cavity after the infection has passed through to the mastoid cavity and has subsided in the middle ear. In these cases the hearing may be only slightly impaired, and the drum membrane may be normal or almost normal in appearance.

Tenderness is probably the most important symptom of acute mastoiditis. It varies with the type of mastoid process. The common sites of tenderness in pneumatic mastoids are over the antrum, tip, premastoid lamina and posterior border. Tenderness is of much less value in the non-cellular type of mastoid process, owing to the very thick cortex.

It is always wise to compare both sides for palpation tenderness and it must be borne in mind that it is possible to elicit tenderness by pressure over the tip in some normal mastoids.

Tenderness may be superficial or deep, generally depending upon whether the mastoid process is cellular or non-cellular.

It is well to remember that tenderness may be present in the early stages of involvement and then subside, but a continuance of tenderness after the acute symptoms have disappeared is quite characteristic of mastoiditis.

The symptoms vary also according to the group, type and virulency of the invading micro-organism.

The most common is the streptococcus; the next most common is the pneumococcus and then follow the Friedlander bacillus, the staphylococcus and others.

In the hemolytic group of streptococci the Pyogenes is probably the most serious offender, as is the Pneumococcus III in the pneumococcic group.

The virulency of any of these invading micro-organisms seems to be increased by a preceding or accompanying infectious disease, such as scarlet fever, measles, influenza, etc.

As a general rule, streptococci produce a severe clinical picture and not much discharge; pneumococci, on the other hand, produce little systemic disturbance and considerable purulent discharge.

Mastoiditis due to the Pneumococcus III is usually very insidious. The onset is similar to any other otitis media and pain is relieved by rupture or incision of the drum membrane, but it is usually followed by an absence of symptoms, except a discharge from the ear and an impairment of hearing. It is often very difficult to persuade the patient that he has a mastoiditis.

Pneumococcus III often attacks old and feeble persons and diabetics; on the other hand, the Pneumococcus III and the bacillus mucosus capsulatus (Friedlander's bacillus) rarely attack infants and young children.

The external auditory canal and the drum membrane present important symptoms and signs of acute mastoiditis, especially in adults.

The rapid accumulation or refilling of the canal with discharge is evidence of the presence of an adjacent larger sized abscess cavity. Pulsation of the discharge as viewed through a speculum, although not diagnostic, is suggestive of a severe inflammation in the mastoid process.

A small perforation situated in the crater of a nipple-like projection of the upper and posterior part of the drum membrane is very suggestive of an acute mastoiditis.

A diminution in the caliber of the canal at the fundus and a prolapse or sagging of the posterior and superior canal wall with no line of demarcation between the canal wall and the drum membrane is pathognomonic of acute mastoiditis. This is due to a periostitis of the canal wall—an extension of the inflammatory process from the adjacent mastoid antrum.

On the other hand, the drum membrane may show no characteristic changes. As stated before, it may be almost normal in appearance in very rare instances, but usually it shows signs of an acute inflammation. In some instances of very virulent infection, the drum membrane may be partially or almost completely destroyed and the middle ear mucosa may be covered with granulations which pour through the perforation.

The diagnosis of acute surgical mastoiditis is usually quite obvious in cases of acute purulent otitis media of several days standing, if one or more of the characteristic signs or symptoms previously mentioned supervene.

The diagnosis may, however, be extremely difficult, particularly in cases of prolonged middle ear suppuration without other manifest symptoms, and in such cases the Roentgenologist and Clinical Pathologist very often give invaluable aid.

4 West 53rd Street.

SYMPOSIUM ON MASTOIDITIS.

DIAGNOSTIC AIDS IN ACUTE MASTOIDITIS: X-RAY.*

DR. FREDERICK M. LAW, New York.

There are two ways in which radiographs of mastoids will help in acute mastoid disease, viz., anatomy and pathology. Their value lies in direct proportion to the extent of your frequency of use and confidence in the interpretation.

Before any interpretation can be attempted, you must be sure you have as technically perfect films as is possible to secure. A poor film is worse than useless; it is misleading.

A perfect film is one in which all bony detail is clearly defined and sharply outlined; one in which the cancellous structure of the temporal bone is seen and the outline of the temporomandibular articulation sharp and distinct. This necessitates the use of a fine focus tube and lack of motion on the part of the patient.

A Bucky diaphragm must not be used. The reasons require considerable explanation and illustration and will be omitted from this paper.

In order to make a comprehensive report the films must be stereo and carefully studied. Now that we have a readable set of films, what help can the surgeon derive from them?

First, and I think foremost, is the knowledge of the anatomy: the size and extent of the mastoid cells and the character of the walls, whether very thin or thickened from previous inflammatory processes. The thickness of the walls determines the possibility of delay in operating or the necessity of early operation. Thick walled cells will naturally resist destruction longer than thin walls.

Is the mastoid well pneumatized or is it non-cellular? If non-cellular, is it undeveloped or is it sclerotic? What is the position

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of the lateral sinus in reference to the posterior canal wall? What is the position of the emissary vein?

If the cell structure extends into the zygoma, does it run in a wide band or does it narrow down into a small channel over the canal to branch out fan-wise as it reaches the glenoid cavity?

Is the tegmen high or low, thick or thin?

Is the petrous bone pneumatized to the apex?

Is there any suspicion of erosion of the apex of the petrous pyramid?

After studying the anatomy we must consider pathology and here is where we must give serious consideration to the quality of the films. The present technique of using intensifying screens and low voltage produces films of greater contrast than we formerly secured from the glass plates or films without intensifying screens, and to those of you who learned interpretation of plates it will be necessary to modify your ideas of the degrees of involvement as registered by the amount of opacity or clouding of the mastoid. We seldom see a mastoid film as opaque as in former years, and in the case of a double mastoiditis one must be cautious in judging the degree of the change. So much detail and contrast is presented by a modern set of mastoid films that one must realize there is more involvement than is indicated by the amount of clouding. More dependence must be placed on the change of detail of the trabeculae. In order to properly interpret these changes, stereo films are a necessity, not a luxury. Viewing a flat film there may not be marked clouding and the cell structure can be very clearly seen, but if we view a pair of stereo films of the same case there may be a total absence of the cell trabeculae in the deeper structures, either around the antrum or between the sinus groove and the posterior canal wall. Thus the stereo films reveal a surgical case while the single film would suggest delay in operation.

Of course, such a condition would be entirely masked by the slightest movement of the head. This means that the head must be securely fastened to prevent movement, and I find that many roentgenologists do not fasten the head during an exposure. Even with an exposure of one-half second there is ample time for movement.

Next we observe the size of the cells. A large cell type will drain easier than a small cell and therefore will be less liable to go on to

necrosis than the small cell type. A small cell type will require a longer time for resolution than a large cell type.

The thickness of the cell walls will indicate whether delay in operative procedure is safe. If the walls are very thin, breaking down will occur sooner than if they are thick, and delay is not wise. I have seen cases in which the walls were thick, bone destruction had occurred, no operation done and radiographs after resolution showed a large cavity corresponding to the area of absorption. Films made three years later showed a complete regeneration of the cell structure. Had this case been one of thin walls, the absorption would have occupied the entire mastoid area.

The cases of acute mastoiditis in which we desire the most information is that of infants, and here is where we often get very little help. In the first place, it is extremely difficult to fix the child's head. Two assistants are unable, at times, to even keep an infant's head under the X-ray tube, and even with an extremely short exposure the resulting film is not satisfactory. The most reliable information obtained is a knowledge of the state of development of the mastoid process. If clear films are obtained (it is next to impossible to secure stereoscopic films) interpretative judgment must rely on the quality and density of the mastoid process in conjunction with the clinical signs.

Then, too, it is practically impossible to keep the auricle folded forward and the outline of the cartilage may simulate an area of bone absorption in an undeveloped mastoid process.

Even where satisfactory films are obtained, a careful study must be made before any opinion is given.

There has been considerable comment recently regarding involvement of the petrous bone and it is wise to make a film of the petrous bone in all cases of acute mastoiditis so that a means of comparison may be had if the case develops symptoms pointing to involvement. There are variations in the appearance of the petrous bones of the two sides in non-involved mastoid cases and a means of comparison is of distinct value when symptoms of this type develop.

Now that we have satisfactory films and an interpretation, a consultation between the surgeon and roentgenologist is advisable in order to correlate the clinical evidence and the radiograph. A consultation may clarify some indefinite appearance on the film or it is possible the clinical findings may influence the roentgenologist in his opinion of the possible outcome.

In closing, let me reduce the diagnostic aid to a mathematical formula:

Let X = the diagnostic aid.

T = technique.

I = interpretation.

C = clinical evidence.

Then to secure the most aid or X, we will have $X = \frac{T + I}{C}$

Keep this formula in mind when considering all radiographs and you will derive the most benefit from this very valuable assistance, and, above all, give the Roentgenologist the benefit of the clinical findings at the time of the examination.

140 East 54th Street.

SYMPOSIUM ON MASTOIDITIS.

DIAGNOSTIC AIDS IN MASTOIDITIS: LABORATORY.*

Dr. Andrew A. Eggston, New York.

What I have to say is from the laboratory viewpoint as a result of my experience as pathologist at the Manhattan Eye, Ear, Nose and Throat Hospital. Laboratory tests reveal the general reactions of the body and are different from the X-ray findings, which are more localizing.

There is little new in the laboratory diagnosis of acute mastoiditis and you probably know the interpretation of the usual laboratory findings. However, I will speak of some of the points that have been impressed upon me as most relevant. Cultures of ear infections are important in order to determine the type of infection to be considered. Not infrequently the first culture will be a mixed one, consisting of staphylococci, streptococci, etc. If the culture is taken from the myringotomy knife, one is more likely to get a pure culture which will furnish valuable information. Infections in acute mastoiditis are usually hemolytic streptococcus pyogenes or streptococcus mucosus capsulatus. This is strange that these two organisms are usually the offenders; I don't know why, but there may be some physical reason on the part of the bacteria which allows them to remain attached to the cells of the mastoid. The other types are usually in pairs or not so large and long and can more easily be removed. At any rate, it is rather interesting that we get these particular organisms more commonly than the other type of pneumococci.

It is important and interesting to compare the organism found early in an ear infection with that in the mastoid or subsequent complications. We formerly typed the different streptococci in a large number of cases, using Holman's classification, based upon the sugar reaction, first placing them in different groups as to the non-hemolytic and the hemolytic characters. The sugar reactions have been discontinued, as very little information of practical value was obtained.

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Non-hemolytic streptococci are rare in the ear, and more rarely in the blood stream. Staphylococci may be found in the mastoid and blood stream, and are usually very serious infections.

Routine urinalysis is, of course, important to determine if a blood chemistry should be done; acute mastoiditis not infrequently occurs in diabetes. We have had several such cases with diabetic coma. If diabetic cases are diagnosed early enough and properly metabolized, they may be operated upon as safely as any other. However, urinalysis does not always furnish proof as to whether or not a patient is a diabetic. There may be sugar in the urine on account of a low renal threshold and the blood sugar be normal. On the other hand, a patient may have a high blood sugar and no sugar in the urine.

Blood counts are utilized as a diagnostic aid in acute mastoiditis to the same effect as in other septic diseases. The changes of the leucocytes are not as abrupt and significant as in acute appendicitis, probably due to the difference in absorptive areas. In a high white count, for example, 25,000 to 30,000, there may be a mastoiditis, but one must consider other septic processes, especially cervical adenitis, pneumonia, meningitis or erysipelas, as an uncomplicated mastoiditis is rarely accompanied by such a high count. One must consider the septic factor of a white count, referring especially to the polynuclear cells of both the mature and immature types. As a rule, an increase in the leucocytes is accompanied by an increase in the polys. With a total leucocyte count of 10,000 and a poly of 70, it shows a normal resistance or fairly mild infection; but if there are 80 or 90 per cent polys, the resistance is poor on the part of the patient, or there is a very severe infection. On the other hand, if there is a high total leucocyte count, with a relative low polynucleosis, the resistance index is high; therefore, a poly decrease reveals better resistance on the part of the patient or a clearing up of the infection. That is true in adults, but in children there are exceptions, because, instead of reacting with polys, they sometimes react with lymphocytes. Another phase of the white cells has lately received a great deal of attention, namely, the young or immature polymorphonuclear leucocytes. A poly whose nuclear lobes are of the younger forms contains less filaments than leucocytes of the mature type. The immature forms are cells with horseshoe shaped nuclei. These immature or unsegmented polys indicate that the bone marrow is being forced to issue more cells to the circulatory blood, which is a response to a septic process and repeated blood

examinations will indicate if the process is progressing poorly or favorably. In acute mastoid disease it is important to repeat the counts at 12 to 24 hour intervals and thus make a comparative chart. Blood counts sometimes reveal a leucopenia. We had one case in which there were only 800 leucocytes and no polys—a typical granulocytopenia. The boy was ten years old and came in with an acute mastoid. By means of repeated transfusions the leucocytes got up to 2,000 with 15% polys. The mastoid was operated upon. The leucocyte count dropped to 1,000, with a relative decrease in the polys. Further transfusions were given and in four to five weeks the blood count was normal. He remained in the ward for his wound to heal, but an acute throat infection occurred and in two days he was dead from granulocytopenia in spite of everything that was done.

Another blood test that is being used in some hospitals is the sedimentation test, but in my experience it has no specific indication. The test consists of mixing the blood with an anticoagulating chemical and noting the time of separation of the cells from the plasma by gravitation. In all diseases, regardless of whether it be tuberculosis, cancer, acute mastoiditis, the rate of sedimentation is increased to a variable degree. The rate of increase, in my opinion, simply indicates the degree of illness of the patient, but has no specific diagnostic value.

Blood cultures are probably the most important of the laboratory procedure in following the complications of mastoiditis. Sometimes it is good policy to culture the blood before a simple mastoidectomy, because the blood cultures may become positive early and when this is true, no time need be lost in obliterating the sinus. Ordinarily a simple mastoidectomy is done and in a few days, if the patient continues septic, it is of extreme importance to determine if the blood stream contains bacteria. A bacteremia following an acute mastoiditis is usually due to a septic lateral sinus thrombosis provided pneumonia or bacterial endocarditis has been eliminated. While a positive blood culture is quite significant of septic sinus thrombosis, a negative one is of limited value. There are several explanations of the variable findings in blood cultures. In the parietal type of thrombus, where the blood continues to circulate and organisms are washed off into the bloodstream, the culture is usually positive. On the other hand, the bloodstream is capable of destroying many bacteria and render itself sterile. If the sinus is completely closed by a blood clot, the flow of blood is entirely interrupted and

the culture is likely to be negative. In cases where the thrombus has become autolysed the bacteria will be thrown into circulation and a large number of bacteria can be secured in the culture. In a review of 450 blood cultures at the Manhattan Eye and Ear Hospital, there were 15.7 per cent positive cultures. These cultures were taken on clinically suspicious cases, not necessarily positive ones. A great many of the cultured cases undoubtedly were not sinus thrombosis, but had some other complications.

The Toby-Ayer's spinal manometer test is of value to determine sinus thrombosis and it is also helpful in determining which side is involved in a bilateral case.

The spinal fluid examinations in mastoid cases are also of importance. It is well to have a lumbar puncture and examination of the spinal fluid in all obscure septic cases following mastoiditis where meningeal symptoms are present. I do not think this procedure harmful, as claimed by some, but is of diagnostic assistance. Outside of the total cell count and the character of the bacteria present, one can get a certain amount of information by studying the character of the cells. If the polymorphonuclears predominate, it is an acute process. If the polymorphonuclear cells decrease and there is found a relative increase in the mononuclear cells, the meningeal process is becoming localized with a probable abscess formation. Other tests are made on the fluid, such as sugar, lactic acid and bacterial cultures. Positive cultures in the spinal fluid are not always obtained, even though bacteria are found by stained films. This is a good prognostic sign, particularly if the pus and bacteria have been concentrated before culturing. If there is a profuse growth of hemolytic streptococcus the prognosis is always bad.

Repeated cell counts of 3,000 or less, with a decreasing cell content, is quite suggestive of the development of brain abscess. In one case, hemolytic streptococci were found on culture of the spinal fluid, which disappeared and in six or seven weeks the patient developed a brain abscess. In an addition to a decrease in number of the cells one should consider degenerative changes in the polys, with an increase in the mononuclears, the process becomes more chronic or is localizing. In such cases the prognosis is better. I place considerable confidence in the cell count and especially the character of the cells. The spinal fluid should be centrifuged in taking cultures.

Cases with frankly turbid fluid and with numerous bacteria are hopeless cases. Nothing we have done has cured a single case of

purulent meningitis. If the cell count is below 3,000, there may be a chance of recovery. Quite a number of such cases have been reported to have recovered even with streptococci in the fluid, but I doubt the existence of an active infective meningeal process in the recovered cases.

After this resumé of the clinical pathology, I wish to refer to our experience with blood transfusions in mastoid disease and its complications. The complications most common to mastoid disease are erysipelas, cervical lymph adenitis, sinus thrombosis, bacteremia, pneumonia and septic meningitis.

In the last 15 years we have been interested in treating the complications of mastoiditis by building up the patient's resistance with blood transfusions. The number of cases studied makes ours a rather unique experience in treating septic conditions in otology by blood transfusions. Any seriously ill patient who must submit to a major operation, complicated by a septic infection, had best have his general resistance increased by blood transfusion. The general surgeons are recognizing the benefits of this preliminary preparation. Mastoid infections so frequently occur after scarlet fever, measles, influenza, diphtheria—all very devitalizing diseases—and there is no place in all medicine where such beneficial results can be obtained from a therapeutic agent as with transfusion, especially in children who have become devitalized by such infections.

653 Park Avenue.

SYMPOSIUM ON MASTOIDITIS.

DIFFERENTIAL DIAGNOSIS IN ACUTE MASTOID DISEASE.*

Dr. Marvin F. Jones, New York.

Differential diagnosis in acute mastoiditis is a limited topic.

Furunculosis is the most common, and may be the most difficult of otological diseases to differentiate from acute mastoiditis. I have seen two normal mastoids operated upon. Both were cases of furunculosis. In one I performed the operation myself. I was an onlooker at the other operation.

Differentiation between acute mastoiditis and furunculosis, uncomplicated, is simple:

FURUNCULOSIS.

1. Direct pressure over the mastoid elicits no tenderness.

2. Manipulation of the auricle, which produces tension on the canal, or pressure on the canal causes pain.

3. On inspection the canal shows an asymmetry.

4. Swelling over the mastoid area which "pits" on pressure. This pressure produces little pain. If the pressure is directed forward, it is painful.

5. Discharge is present only if the furuncle has ruptured. The character is thick, but not particularly profuse. The source of the discharge is from the canal wall.

6. Temperature and blood picture are nearer normal.

7. Roentgenograms may show clouding over the mastoid area but no cell destruction.

ACUTE MASTOIDITIS.

1. Direct pressure over the mastoid elicits tenderness.

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2. Manipulation of the auricle or pressure on the canal is not painful.

3. The canal may be constricted but not distorted.

4. When complicated by sub-periosteal abscess it exhibits swelling but without the marked "pitting" and is very tender.

5. Discharge is present if the drum is open and usually is profuse. The source of the discharge is from behind the drum.

6. Temperature elevated with blood picture shows sepsis and sometimes diminishing red cells.

7. Roentgenograms show clouding plus destruction of cells.

Less obvious pictures are presented when a combination of furunculosis and mastoiditis are present.

I have seen cases with a grossly normal drum with a swelling of the posterior canal wall resembling in all respects a furuncle. Rupture and drainage was not followed by a speedy resolution. Further trouble was suspected and found in the mastoid. The explanation advanced was that the pus had eroded the posterior canal wall and ruptured into the canal.

This is a situation similar to the one we find in sub-periosteal abscess and Bezold perforation, the difference being in the escaping point of pus from the mastoid cells.

The existence of furunculosis super-imposed upon an acute exacerbation of a chronic mastoiditis presents a real problem. Probably our greatest help comes from satisfactory roentgenograms. However, the roentgenograms do not always help us out of our difficulty, especially if the mastoid happens to be of the infantile type, or sclerotic. These cases tax the ability and ingenuity of the otologist and must be handled as individual problems.

Post-auricular lymph nodes sometimes enlarge, are tender and suppurate. Negative fundus, negative roentgenograms and history usually make the differential diagnosis easy.

Pain in and around the ear immediately suggests mastoiditis to our patients. We are therefore faced with a problem which may be considered under differential diagnosis. This symptom is placed in the class of referred pain. The seriousness of this situation was emphasized after seeing two cases which had been prepared for mastoid operations. Both cases had infantile types of mastoids as re-

ported by the roentgenologist. The cases were supposed to be that rather rare condition known as primary mastoiditis. The dentist cured both cases.

The most common sources of these referred pains are the teeth, sinuses, tonsils and nasopharynx.

Dental pathology produces pain which is referred to the area in and around the ear. Dental cases with inflamed pulp and exposed nerves, abscesses and bone necrosis around the roots constitute some of these pathological conditions.

Involvement of the sphenoid sinus and posterior ethmoid cells produces a dull aching sensation which is vaguely located back of the ear and over the occiput. Pain referred from the antrum has expressed itself in a more acute and more localized form. Frontal sinuses with supra-orbital pain seem to radiate to the ear. The pain referred from the tonsils is the result of an acute tonsillitis, peritonsillar abscess or from a post-operative tonsillar fossa. This pain is severe at times and seems to be located in and around the middle ear.

The nasopharynx and Eustachian tube may have a raw, dry, granular appearance and when present the patient complains of a sharp stab or lancinating pain in the ear. These pains are usually transient.

Neuralgia or neuritis, especially if the sphenopalatine ganglion be involved, produces auricular pain.

In the absence of aural discharge all these conditions may be differentiated from acute mastoiditis by careful aural examination plus the roentgenogram.

When discharge is present in the canal plus referred pain the operability of the mastoid may be in question. A sequential history, the character of the discharge, appearance of the fundus, lack of mastoid tenderness, normal blood picture, normal temperature and the roentgenogram form the basis for an opinion which eliminates the mastoid as a cause of the pain.

The lay public and I might add, some of our profession, have developed a disproportionate fear of mastoiditis. In a psychoneurotic individual this fear sometimes assumes a very real form. He develops the characteristic symptoms of an acute mastoid. I have seen some of these cases in the Neurological Clinic at the Post Graduate Hospital, and the differential diagnosis between an hys-

terical and a real mastoid may be puzzling. The middle ear in these cases may or may not be normal. A characteristic feature of these cases is an extreme hypersensitiveness and an aversion to examination. Stroking the finger lightly over the mastoid area will make these patients pull away and scream with pain. If the confidence of these patients can be gained and their attention diverted, pressure may be exerted without discomfort.

Eczema, external otitis diffuse, erysipelas, malignancy, perichondritis and trauma may be mentioned as diseases which might be mistaken for mastoiditis. They are so commonly self-evident that their consideration is omitted.

In closing, I wish to note the value of roentgenograms in differential diagnosis. We are misled at times by the opinions of the best roentgenologists on the best of pictures. We are often misled by roentgenologists who have little knowledge of head radiography by their opinion on poor pictures. I can heartily subscribe to the opinion of Ross Skillern which has been expressed on numerous occasions, *i. e.*, every otolaryngologist should develop the ability to interpret roentgenograms of the head. He should make his clinical diagnosis and then examine the roentgenograms. From the two his own diagnosis is made. He should then read the interpretation given him by the best roentgenologist obtainable and check against his own conclusions. Certainly by this procedure the roentgenograms would be made a very useful aid in differential diagnosis.

121 East 60th Street.

THE EVOLUTION OF THE MASTOID TIP CELL AS A CELL SYSTEM SEPARATE FROM THE REMAINDER OF THE MASTOID CELLS, AND ITS SIGNIFICANCE (PRELIMINARY REPORT).*

DR. RALPH ALMOUR, New York.

The investigation by Wittmaack into the process of pneumatization of the temporal bone has thrown a great deal of added information upon the development and course of otitic infections.

The middle ear and antrum at birth are filled with embryonal subepithelial tissue which, in the normal process of development, grows into the marrow spaces of the mastoid process and replaces the marrow contained between the bony trabeculae. When this myxomatous tissue undergoes its normal contraction into the adult type of connective tissue, it causes the epithelial lining to be drawn into the intertrabecular spaces until a series of air cells results which communicate with the middle ear from the antrum.

We have been taught that the tympanic antrum is the mother cell from which all other mastoid cells develop. However, on histological examination of infants' temporal bones there can be demonstrated an offshoot of this subepithelial tissue which spreads toward the mastoid process underneath the descending fallopian canal. This offshoot is entirely separate and distinct from that located in the tympanic antrum. It takes its origin from the hypotympanum at the point where the posterior and inferior tympanic walls meet. It then goes backward and downward to enter the mastoid process. Wittmaack[1] observed this and believed that this was a separate process concerned in the pneumatization of the tip.

If an adult temporal bone is examined, a definite tract can be found leading from the middle ear into the tip cell, and this can be demonstrated in the following manner:

A well pneumatized adult bone should be selected. The thin cortex should be shaved off, preferably with a fine chisel, thus bringing into view the superficial layer of the mastoid cells. A syringe filled with water is now inserted into the bony external auditory

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meatus so that the tip fits snugly into the canal and no leakage occurs around it. If the water is now injected, it will be seen to come out of the mastoid process through all the cells. If the outer attic wall is now removed with a fine drill and the epitympanic space and aditus are brought into full view through the external auditory canal, and if the opening to the antrum is now plugged off, using either modelling clay or even chewing gum, and we again inject through the external auditory canal in the same manner as before, the water will be seen to trickle out only from the cells at the tip.

If this same bone is now thoroughly dried and a coloring solution, such as gentian violet or methylene blue, is injected into the external auditory meatus, exerting great care not to have any leakage, the tract from the middle ear into the tip cell will be outlined and can be dissected. Starting at the mastoid tip, this tract is found to follow from the tip cell upward and inward along the posterior canal wall and then directly inward, overlying the lateral sinus, until it reaches an area located posterior to the jugular bulb. This tract then runs underneath the facial nerve to emerge in the middle ear at the innermost part of the junction between the posterior and inferior tympanic walls.

This tract can also be demonstrated in another manner. A fresh adult temporal bone should be obtained. The soft tissues should be removed with scissors. The anterior and inferior walls of the external auditory canal are next removed and the hypotympanum brought into full view. Using a fine stiff wire, search is now made along the posterior wall at its junction with the inferior tympanic wall until the probe is found to enter a cavity. This point will be found to be situated almost perpendicularly downward from the round window. With this probe in situ, the dissection of the mastoid process can next be carried out, starting from the tip, until the probe is brought into view through the exposure made in the mastoid process.

This work is based upon the dissection of and experimentation with twenty-four well pneumatized temporal bones. In all of these, the tract from the middle ear to the tip cell was demonstrable. It is conceivable that the tip cell may be pneumatized while the remainder of the mastoid process is sclerotic or diploic. This has not yet been investigated.

This additional pathway from the middle ear into the mastoid process is of significance in that it tells us that infection can spread

by contiguity from the middle ear into the mastoid cells without spreading from the antrum. It explains for us the early appearance of tenderness over the mastoid tip in cases of acute purulent otitis media. This point of tenderness may be the only one present during the entire course of the disease. It also explains for us the frequent finding on roentgen examination of intact cells in the region of the antrum and throughout the major portion of the mastoid process, in the presence of exquisite tip tenderness. It also accounts for the frequent finding on the operating table of isolated empyema of the mastoid tip cells with intact cells through the remainder of the mastoid process. It may also be a factor accounting for the presence of early facial paralysis in infants, since the pathway into the tip from the middle ear surrounds the facial canal. It may possibly account for the early involvement of the jugular bulb by a thrombus.

Further studies along clinical lines are necessary before the full significance of this pathway from the middle ear into the tip cells can be estimated. Up to the present time the finding of pus within the mastoid process was considered indicative of an acute mastoiditis. With closer observation at the operating table, correlated with close study of the roentgen plates, it may be found that these isolated empyemas of the mastoid tip cells can be cured by drainage of the tip, exposure of the peribulbar cells and opening of the secondary antrum, without complete exenteration of the remainder of the mastoid cells.

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51 West 73rd Street.

AN AID TO INTERPRETATION OF INTRACRANIAL COMPLICATIONS RESULTING FROM VENOUS CIRCULATORY DISTURBANCE OF THE TEMPORAL BONE, OFFERED BY X-RAY OF THE LATERAL SINUS AND JUGULAR FORAMEN.*†

DR. MATTHEW S. ERSNER and DR. DAVID MYERS,
Philadelphia.

It is a well known fact that venous circulatory disturbance of otitic origin produces intracranial pressure changes. It has also been observed that meningeal symptoms will manifest themselves under these conditions. The latter depend upon the severity of the infection, type of temporal and mastoid bone and the state of the venous circulation.

We will endeavor to identify some of these disturbances as being due primarily to anomalies of the lateral sinus and jugular foramen and possibly other vessels not yet visualized by the X-ray.

In perusing the otologic literature, we did not encounter any X-ray illustrations depicting circulatory anomalies. It is therefore our purpose to demonstrate and visualize these structures via the Roentgen ray and substantiate these findings by clinical observations.

It is true that the X-ray study of the temporal bone is indispensable and until recently all X-ray studies were centered about the mastoid process, where we were enabled to ascertain the following:

1. Comparative study between the normal and diseased mastoid.

2. Type of mastoid, whether pneumatic, diploic or sclerotic.

3. Topographical and regional anatomy of the mastoid, its cellular distribution, type of cells, position of the lateral sinus and its surrounding area.

4. In atypical mastoiditis, the X-ray is indispensable as an aid in diagnosis.[1]

5. Exudative mastoiditis.

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*Read before the Philadelphia Laryngological Society, Philadelphia, Pa., April 4, 1933.

†From the Department of Otology, School of Medicine, Temple University.

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6. Bony absorption.
7. Cavity formation.
8. Emissary vein.
9. Diagnosis of perisinus abscess.
10. Epidural abscess.
11. Bilateral suppurative mastoiditis.
12. Resolution.
13. Visualization of the petrous portion of the temporal bone.

Today, however, we can go a step further and demonstrate by the aid of the X-ray the size of the lateral sinuses and jugular foramina. This has been of invaluable aid to us.

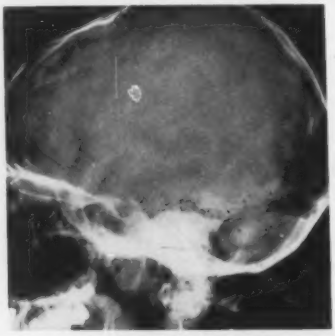

Fig. 1.

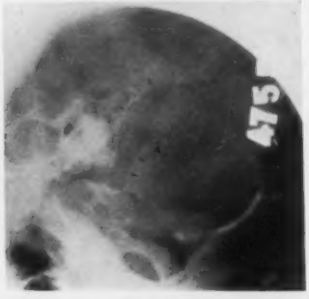

Fig. 2.

Fig. 1. Flat plate showing left lateral sinus.

Fig. 2. Lateral sinus beginning at torcular herophili and terminating in jugular bulb. Letter (A) indicates the emissary vein entering the lateral sinus.

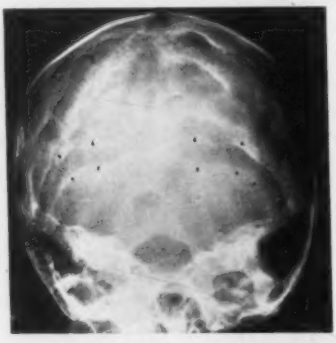

Fig. 3. Coronal view, illustrating lateral sinus. This view is best for comparative study as to size of the lateral sinuses. (These films are usually viewed through the stereoscope.)

In otologic discussions the location of the lateral sinus is quoted frequently as being of certain strategic importance to the operator. To our minds it is of least significance. It is the purpose of this paper to stress the importance of the size of the lateral sinuses and the jugular foramina and of comparing the difference in each side. We believe that knowledge of this disproportion is of great prognostic value and often aids in the interpretation of various neurological symptoms that occur as the result of disturbance in the intracranial venous circulation.

It has long been known that the right lateral sinus is larger than the left. Tobey[2] in his discussion of the Queckenstedt test as it relates to lateral sinus thrombosis, states, "Anatomic variations in the size of the lateral sinus must be admitted, although a demonstrable difference in the size of the two lateral sinuses is unusual."

Due to improved X-ray technique (method developed by Dr. W. E. Chamberlain, of Temple University Hospital, Department of Radiology), we are now in a position to demonstrate the difference in the two lateral sinuses and also the jugular foramina. We emphasize this so that it should become a routine procedure.

Our reason for recommending this procedure is due to the fact that we carefully correlated the X-ray findings, clinical picture and operative findings in many cases and we feel that we can often prognosticate and interpret various neurological phenomena.

At this time a short review of the anatomy of the cranial sinuses would not be amiss. Notwithstanding the fact that this is common knowledge we have included a short outline abstrated by Seydell.[6]

1. Superior longitudinal sinus.

2. Lateral sinus—horizontal, sigmoid.

a. Superior petrosal empties into the sigmoid at the base of the petrous portion of the temporal bone.

b. Mastoid emissary joins the lateral sinus at a point slightly below the opening of the superior petrosal sinus and emerges through the mastoid foramen, on the external surface this is found just behind the posterior limit of the mastoid process.

c. Anterior and posterior condylar veins, which empty into the sigmoid sinus at the junction between the sigmoid sinus and the bulb.

d. Inferior cerebellar and cerebral veins.

e. Petrosquamous sinus frequently absent, runs along the petrosquamous suture.

f. Inferior petrosal sinus, which joins the sigmoid sinus where the latter rests on the jugular process of the occipital bone. The junction of the two forms the beginning of the internal jugular vein. This drains the tympanum, cochlea, internal ear and aqueduct.

g. Inferior longitudinal sinus situated in free margin of the falix cerebri and usually empties into the left lateral sinus.

h. Occipital sinus begins at the foramen magnum, and communicates with the posterior spinal veins and terminates in the torcular.

i. The torcular herophili is located on the internal occipital protuberance and is formed by the junction of the superior longitudinal, straight, lateral and occipital sinuses.

Anatomical variations in the size of the jugular foramen are mentioned by Linser, quoted by Seydell.[4] "Linser examined 1022 skulls and found that 3 per cent had an extremely small jugular foramen on one side. This finding occurred three times more frequently on the left side than on the right. The foramen, in these cases, was too small to allow the passage of a vein that could establish collateral circulation and undoubtedly severe reactions would have resulted in these cases, and perhaps death might have occurred had ligation been restorted to."

E. H. Campbell[5] quotes Don Campbell[6] and states that statistics show that the right lateral sinus is larger than the left and that its relationship to the mastoid cells is such that it is rendered more liable to infection than the left in the proportions of 3:2.

Kopetzky[7] quoted by Law, who before the Austrian Otological Society presented an anatomic specimen of a right temporal bone which showed a strongly developed emissary vein, running upward and posteriorly in a sulcus 6.5 mm. in width. The sulcus of the sigmoid was entirely absent, the blood from the right transverse sinus emptied into the right emissary vein and not into the jugular bulb. From the emissary, the communication was with the superficial cervical veins directly. The left sigmoid sinus was markedly developed. In addition the right middle and posterior cranial fossae were smaller than the left. He quoted Frey and Hoffman as having presented a similar observation in 1924.

Very recently (March 20, 1933) we had the opportunity of operating on a patient, S. K., male, age 5 years, for an acute right

mastoiditis (see Figs. 4 and 5). Following our routine, X-ray of the jugular foramina and lateral sinuses was done. We were able to demonstrate the absence of the lateral sinus and the presence of a large emissary vein on the right side, preoperatively. These findings were confirmed at operation. The left side showed a tremendous left lateral sinus and jugular foramina, three to four times the normal size by X-ray. At operation we exposed the large emissary on

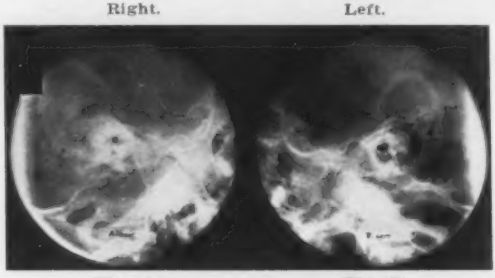

Fig. 4A. Fig. 4B.

Fig. 4. (A) Shows absence of right lateral sinus and presence of a large emissary vein. (B) Shows the tremendously large left lateral sinus.

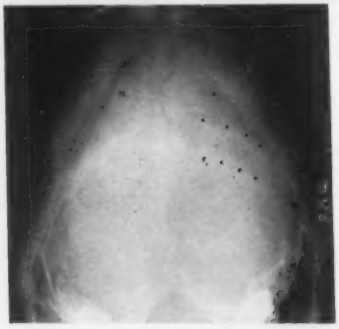

Fig. 5. Coronal view of same patient shows the large left external sinus, and the absence of the right lateral sinus.

the right side and a portion of the cerebellar dura and we were able to follow it to its point of entrance into the jugular bulb; no evidence of a lateral sinus was noted.

Ballance,[8] in 1890, wrote as follows: "When the internal jugular vein is tied, the face may become blue on consequence of the obstruction to venous circulation. The right internal jugular vein is usually a little larger than the left, since it is continuous with the right lateral sinus, which is generally more capacious than the left.

The venous turgescence would probably therefore be more marked in cases in which it was necessary to interfere on the right side.

"Sometimes the lateral sinus is much smaller on one side than on the other. In the museum of St. Thomas' Hospital there is a skull which has no groove in it on one side for the lateral sinus. So, in this case, during life, on the side in which the groove was absent the lateral sinus must also have been wanting, or only represented by a very thin channel in the dura mater. It thus appears that a man may get along very well with only one lateral sinus."

Dr. George M. Coates has a specimen of a temporal bone in which the left lateral sinus is totally absent.

Cunningham states "that the internal jugular vein is sometimes smaller or larger than normal. In either case compensating changes in size occur in the lateral sinus and the internal jugular vein of the opposite side, or in the internal or anterior jugular of the same side. One lateral sinus may be absent or very small."

In our experience, we have encountered three instances where the lateral sinus was absent, and in another case, although the lateral sinus was present, it was very small.

Further evidences showing the difference in the size of the lateral sinus have been shown by Eagleton,[9] in 1906.

He quoted Knott, McEwen, Komer, Street, Panse, Kasloff and Dumont, Bankow, Biddle and Zuckerkandl as having recorded de-

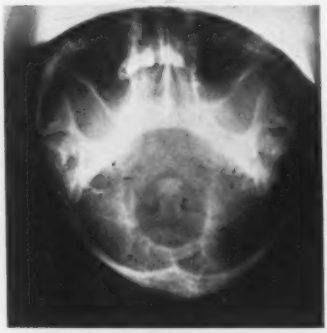

Fig. 6.

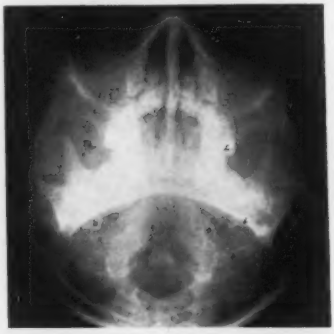

Fig. 7.

Fig. 6. X-ray of jugular foramina. Right side is of normal size, and left side is very small (semilunar).

Fig. 7. X-ray of jugular foramina, illustrates the left side as much larger than the normal with practically an absence of the right jugular foramina. This is the case illustrated in Figs. 4 and 5, where there was an absence of the lateral sinus on the right with the presence of a large emissary vein.

monstrable anatomical differences or absence of the lateral sinuses and jugular foramen.

Zuckerkandl found a right jugular foramen almost eight times the size of the left; Biddle found a contracted left jugular foramen, and the right was so small that a bristle could hardly be passed through it, but the right mastoid foramen was 1 cm. in size. In most of the cases in which the jugular foramen is small, the mastoid foramen is large and in a few days the transverse sinus goes through it.

The above quoted literature definitely indicates that there is a marked difference in the size of the lateral sinuses and the jugular foramina. These statistics have been gathered from anatomical studies.

To our knowledge, however, after a careful survey of the otologic literature we were unable to find reference where the differences in the size of the lateral sinus and the jugular foramen has been demonstrated by X-ray. Here the X-ray has been utilized on the living to the fullest extent of its clinical importance.

Wanamaker,[10] however, in discussing lateral sinus thrombosis mentions a case which presented marked signs of intracranial pressure, and states that X-ray revealed a small contracted left lateral sinus.

Our purpose in quoting the above literature is to substantiate our observations and X-ray findings that there are demonstrable venous circulatory differences. It is our aim to apply these findings clinically, and to utilize them in the diagnosis, treatment and prognosis of intracranial complications; vis., sinus phlebitis, thrombosis, Gradenigo's syndrome, serous meningitis or the otitic hydrocephalus of Symonds[11] and certain types of petrositis and osteomyelitis of the petron, presenting clinical and X-ray findings.

Before presenting a detailed discussion of each of the above conditions, we wish to call attention to some of the mechanical and pathological phenomena due to disturbed venous circulation.

Symptoms of circulatory disturbance due to venous congestion may manifest themselves by nausea, vomiting, edema of superficial vessels of the scalp and papillitis. Then there may be gradations from the simple to the most complex pictures with manifestations of ocular palsies, such as external rectus, et cetera, and further degrees of optic neuritis, choked disc, various meningeal symptoms, as Kernig's sign, Brudzinski, rigidity of the neck, and increased

spinal fluid pressure. These symptoms are indicative of an increase of intracranial pressure. This appears to be the natural outcome of an increased intravenous pressure, since the changes in the mechanical pressure within the cerebral veins must influence directly the pressure in the surrounding cerebrospinal fluid.[12]

Most observers who have measured this intracranial pressure say that it is the same as the venous pressure within the sinuses. This we can understand when we remember the close relationship existing between the subarachnoid fluid and the larger veins and sinuses. An increase in venous pressure might be assumed to cause a corresponding rise in intracranial pressure due to compression following the expansion of the venous walls, and to the retardation of the inflow of cerebrospinal fluid into the veins.[13]

"Changes in vascular volume can also be compensated within certain limits at the expense of the cerebrospinal fluid bulk, as can readily be shown by forcing out of spinal fluid when jugular pressure is applied in the Queckenstedt test. This test, however, because of its abrupt application causes a rise in intracranial pressure sometimes to over 40 mm. of mercury, and even a slight constriction of the jugular outflow may show a fluctuation of pressure, as demonstrated by Ayer and elaborated by Stookey."[14]

In the ordinary case of simple mastoiditis, intracranial symptoms do not occur. If they do, they are of such a mild character that they pass unnoticed. The problem confronting us is to explain their presence when they do occur.

The factors concerned in the production of these changes in venous pressure are as follows:

1. Mechanical: *a.* Anomalies of venous structures; *b.* size of lateral sinuses; *c.* size of jugular foramina.

2. Pathological changes resulting from the infection.

3. Virulence of the organism.

The mechanical phase dealing with the blood vessel anomalies and the size of the lateral sinus and jugular foramina have been described.

The extent of the pathology depends upon the type of organism, the resistance of the patient, the type of temporal bone and the size of the vessel.

There are several changes that occur in the vessel as the result of inflammatory reactions. The first is a primary and evanescent

constriction of the vessels. This is followed by a more lasting dilatation. The increased rate of blood flow persists for only a short time, and is replaced by a slowing of the current, which may progress to complete stagnation. The slowing of the blood current has been attributed to various influences, including alteration of the viscosity of the blood, swelling of the vascular endothelium, an obscure increase in the thickness and diminution in smoothness of the vessel lining. Wooley, after a careful study of the literature on this subject, comes to the conclusion that "decrease in the rapidity of the blood stream is the result of two factors; namely, an increase in the volume of the cells of the blood and of the endothelial cells lining the blood vessels, and increased viscosity of the blood as a whole. The evidence to support these conclusions is abundant and important."[15]

Resolution is favored in these circulatory disturbances when the contralateral circulation is adequate. Otherwise a series of clinical symptoms appear that indicate the presence of increased intracranial pressure. If the venous congestion continues, there is eventually a damming back of the circulation into the petrosals and the cavernous sinus and the other vessels, resulting in a reverse circulatory current which will eventually extend to the veins of the opposite side. The contralateral circulation therefore becomes the structure which will bear the brunt of carrying on the circulation for both sides. If it so happens that the smaller side must bear the burden, then symptoms significant of intracranial pressure appear. We may venture the opinion that the reason why catastrophes do not occur is because the venous structures yield to increased blood volume which temporarily relieves the situation. Unfortunately, however, these venous vessels must eventually leave the skull, and in doing so pass through their respective bony foramina. These bony foramina are rigid and do not yield as the blood vessels do, and it is then that the train of symptoms progress and catastrophes may occur.

We have numerous cases and X-ray films to substantiate this statement. In the cases that involved the side of the smaller sinus and jugular foramen, the pressure symptoms were practically nil, even those where X-ray revealed a suppuration of the petrous portion of the temporal bone. Those symptoms were absent, due to adequate circulation. Those cases which involved the side of the larger sinus presented the neurologic signs which we recognize as being due to disturbance in venous circulation and increased intracranial pressure. This paradox of the larger vessel being most often involved and presenting complications was difficult to fathom. How-

ever, now we feel that we can explain this as resulting from faulty collateral circulation.

Various authors call our attention to changes in intracranial venous circulation. Ballance,[16] previously referred to, noted the signs of increased intracranial pressure, when the larger blood sinus was involved, and the opposite side is too small to care for the extra load thrown upon it. Seydell[17] states as follows: "More dangerous than metastasis is the severe reactions resulting from the lack of collateral circulation following ligation."

Dr. Eagleton,[18] in 1906, describes with great clarity the intracranial changes as the result of venous disturbance, especially when the aural pathology involved the side of the larger blood sinus.

One of us (M. S. E.), in an extensive otologic practice extending over the past twenty years, has noticed the frequency with which otogenic complications occurred on the right side. It is this relationship between the fact that the right lateral sinus is larger and the fact that the dangerous complications occur more often on the right side, that we are endeavoring to point out.

The rate of involvement of the right lateral sinus to the left lateral sinus may be called to our attention by the following statistics:

Fraser,[19] in a report of twenty-eight cases, found sixteen cases on the right side and twelve cases on the left side. Atkins,[20] in a report of forty cases of lateral sinus thrombosis, found that 85 per cent had right side involvement.

In a review of the literature reported by fifty-seven authors, we collected 175 cases of sinus thrombosis. Of this number, 102, or 58.2 per cent, occurred on the right side and fifty-eight cases, or 33.1 per cent, on the left side, and in fifteen cases, or 8.5 per cent, the side was not mentioned. This is practically a 2:1 ratio.

The various factors influencing a sinus thrombosis are the type of organism, severity of infection, blood stream involvement, et cetera. We will not consider these factors at this time. We wish to emphasize the symptoms that result due to disturbance in the circulation. These are, namely, *1*. Greisinger's sign; *2*. optic neuritis up to choked disc; *3*. Tobey-Ayer test; *4*. Crowe Beck sign; 5. suspicious meningismus or meningitis.

1. Greisinger's sign is described as an edematous swelling over the mastoid, due to engorgement of the emissary vein and is an

indication of the damming back of the circulation from the sigmoid due to thrombosis.

2. Optic neuritis or papillitis: One of the most frequent indications of increased intracranial pressure is the presence of optic neuritis or choked discs.

Eagleton states, "the second important symptom of venous storing was the rapid appearance of choked discs immediately after ligation." Since Kipp directed attention to the importance of optic neuritis as an indication for opening the mastoid, the condition of the fundus has been carefully observed in cases of suppurative otitis media, nevertheless it is only recently that the experiments of Cushing and Kocher have demonstrated choked discs to be due to increased intracranial pressure, accompanied by an obstruction of the venous blood outflow. In these experiments, the effect of regulated mechanical pressure on the cerebral circulation was directly seen through a glass window in the head of the animal operated upon.

The circulatory disturbances were, first, with slightly increased intracranial pressure, a narrowing of the venous channels. During this stage the symptoms in the main were insignificant. If the pressure was further increased, the venous narrowing gave place to the venous stasis and hyperemia of the cerebral veins. This was associated with choked disc and symptoms of cerebral irritation, headache, vertigo, restlessness, et cetera.

Clinically we have exactly this condition produced by occlusion of the sinus by a thrombus, or the ligation of the jugular, if the other venous channels are not large enough to take care of the outflow. This explains the varying degrees of the frequency of optic neuritis in the different pathological lesions, its frequency in sinus thrombosis both before and after operation, in brain tumor, abscess and its infrequency in meningitis, in all of which latter conditions the intracranial pressure must be raised to a high degree in order to produce a vascular condition, which is the primary one in sinus thrombosis. That this is the correct view is further proved by the infrequency of optic neuritis in cavernous sinus thrombosis, where while there is much greater local storing of blood, there is much less cerebral storing. In one case of cavernous sinus thrombosis observed, no optic neuritis occurred."

In view of these facts, we think that it is fair to infer that in all cases of sinus thrombosis, the presence of optic neuritis should warn us that the return flow is already seriously obstructed, and

cause us to be exceptionally careful to add as little further obstruction as possible by our surgical procedure.

Theoretically there are certain conditions possible in sinus thrombosis which may make the ligation of the jugular dangerous by preventing the return circulation, viz., in reference to choked disc, which we have mentioned, it is a definite sign of increased intracranial pressure, and should be viewed in this light, both in relation to diagnosis and prognosis of intracranial complications.

Wilkinson[21] states that optic neuritis is present in 25 per cent of cases of sinus thrombosis.

Greuning[22] states, that "choked disc has an initial, an intermediate and an advanced stage, every one of which is ophthalmoscopically visible, and evidences increased intracranial pressure."

G. L. Tobey[23] states, that optic neuritis is present in only 10 to 11 per cent of cases.

Blau, quoted by Wanamaker,[24] states that "the changes in the eye ground are apparent in one out of five cases."

In our series of sinus thrombosis, we would like to quote the following case:

Patient W. F., male, age 8 years, had a sinus phlebitis involving the right side (see Fig. 8). X-ray revealed a larger left lateral sinus. In this case, no evidence of alterations in the eye ground of the right side occurred, even though the right lateral sinus was obliterated.

This is in agreement with the statement of Benedict:[25] "In a case of complete thrombosis of one lateral or sigmoid sinus the mechanical obstruction to the flow of blood may not produce characteristic changes in the eye."

The reason that the percentage of choked disc varies so, as reported in the literature, is no doubt due to the fact that where the circulation of the opposite side was adequate, this does not occur.

In another case, patient M. H. (see Fig. 9), age 14 years, female, the right side was involved and, incidentally, the larger side, a choked disc of 3 diopters occurred. This persisted for six weeks.

With the knowledge that choked discs may occur in surgical disease of the mastoid process with or without thrombosis of the lateral sinuses, the only conclusion one can reach regarding the sig-

nificance of choked disc is that some intracranial complication has taken place, but the nature of this involvement is not manifest. Probably the gravity of the situation is heightened if choked disc follows septic sinus phlebitis.[26]

The explanation is that the pathology is on the side of the larger sinus and, as a result, the collateral circulation is inadequate and the prognosis in these cases is to be guarded. Cases in which choked discs do not occur are more apt to get well; the reason is now obvious.

Where one finds that there is a small jugular with probably a disturbance of the lateral sinus, he should always look for eye symptoms in the form of papillitis. In these cases in order to avoid trou-

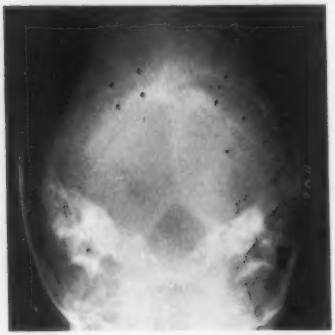

Fig. 8.

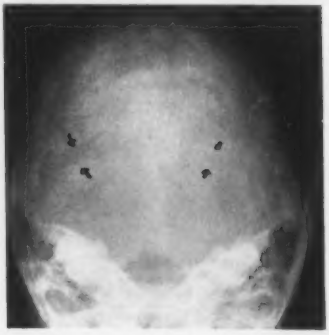

Fig. 9.

Fig. 8. Patient W. F., case of right sinus phlebitis with obliteration of the right lateral sinus. Note the small right lateral sinus and the large left lateral sinus. Eye grounds remained normal.

Fig. 9. Patient M. H. Note presence of large right lateral sinus, and the small left lateral sinus. Sinus thrombosis on right side, choked disc of three diopters due to inadequate contralateral circulation.

ble, one should do a spinal puncture, or use glucose intravenously, magnesium sulphate by bowel, or mouth. This will aid in reducing the intracranial pressure.

Kopetzky[27] has said, "Due to the disturbed, intracranial venous circulation, an increase in amount of cerebrospinal fluid is usually present in sinus thrombosis, due to the interference and the disarrangement of the intracranial circulation."

The Queckenstedt test as utilized by Tobey and Ayer[28] has been considered of great importance in the diagnosis of sinus thrombosis.

We wish to take this opportunity to make several observations contradictory to these findings and disclose several facts that will tend to more than minimize its importance. We believe that the Tobey-Ayer test is only of value when there is a rise of spinal pressure following jugular compression (negative Tobey-Ayer test). Otherwise, if on jugular compression there is no rise in spinal pressure, even in cases of suspected sinus thrombosis, this should not be considered as positive for the presence of a sinus thrombus, for the following reasons:

1. Difference in size of the venous structure.

2. Inability to compress the jugular alone when doing this test because the jugular lies in the carotid sheath.

3. Effect of the carotid sinus reflex, demonstrated by Heyman, Schmidt,[29] Hill,[30] and others.

Further data on this subject is now in the process of publication and will appear later in one of the medical journals.

That disturbances resulting in sinus thrombosis are due in a great measure to the lack of collateral circulation, when the side involved is smaller may be further illustrated in another fashion by the work of Ottenberg.[31] He shows that when blood cultures are taken from the internal jugular vein of the involved side (in sinus thrombosis) and from the opposite internal jugular, there is a greater number of organisms present on the opposite side. This indicates the importance of the contralateral circulation.

Otitis Media with Meningeal Symptoms: A group of patients presented gastrointestinal symptoms in conjunction with meningeal disturbances. Often the meningeal symptoms may be alone and most prominent. We have observed that these may occur either in unilateral or bilateral otitis media. The symptoms invariably disappear after myringotomy. We noted a peculiar coincidence; that there is profuse bleeding, dark bluish in color, no doubt venous in character, following the incision of the eardrum. We attribute the disappearance of this symptom-complex to the fact that we are practically doing a phlebotomy, decongesting the middle ear and the venous supply of the temporal bone. These conditions occur most often on the side where the vessels are larger. Since the right side is larger in 89 per cent of the cases, and we have noted that this type of case does occur more frequently on the right side, then is a right myringotomy more of an emergency than the left? This observation was also noted by Simon L. Ruskin.

Gradenigo's triad was at one time considered a distinct classical entity, and a great deal of stress was laid upon its appearance. Today it remains a syndrome due to the fact that we are unable to explain its occurrence upon an anatomical and pathological basis. Having observed many cases presenting this syndrome and checking upon our X-rays, we would like to suggest that circulatory disturbances are often responsible for this group of symptoms. We have several X-ray films presenting exudative processes in the petrous portion of the temporal bone, this occurring on the side of the larger blood vessels. On the other hand, we have X-rays of exudative conditions in the petron on the smaller side. Under these conditions the Gradenigo syndrome is absent.

Petrositis occurring on the side with the larger blood vessel is

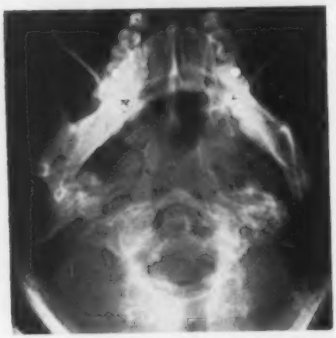

Fig. 10.

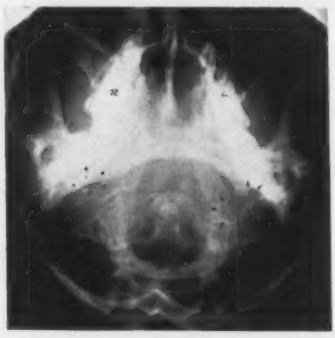

Fig. 11.

Fig. 10. Patient K. McG. X-ray evidence of petrositis on the right side. Deep seated pain in the right eye was constant. Recovery following extensive right simple mastoidectomy.

Fig. 11. Patient K. McG. Note large left jugular foramina. Collateral circulation adequate, an important factor in an uneventful recovery.

apt to have involvement of the sixth nerve due to vasostasis and faulty contralateral circulation.

It is not our thought to be biased on the subject in order to prove our point, but we have concrete data showing that in three cases, where X-ray studies were made, suppurative changes took place but the circulatory disproportion was such that the larger vessel was on the healthy side (see Fig. 11). Three cases recovered, and one case, where the larger side was involved, succumbed. We therefore feel that the recoveries here were due to the adequate circu'ation and the circulatory system was of such nature that collateral circulation

became re-established. Therefore, we feel justified in our attitude by not resorting to radical surgery on the petrous portion of the temporal bone. None of these patients, except the one who died, showed any papillitis or choked disc.

Otitic hydrocephalus is a condition which often appears concomitant with otitis media and mastoiditis. The symptoms depend upon the amount of cerebrospinal fluid and the pressure exerted upon the nervous system. Occasionally, there are isolated areas of fluid collections. The symptoms are therefore not constant, due to the above factors. In the early portion of this paper we called attention to the close relationship between the venous circulation and the cerebrospinal fluid and that these structures are interdependent upon one another. The physiologists tell us that the spinal fluid is produced through the choroid plexus and is absorbed by the pacchionian bodies. Circulatory disturbances with increased localized, as well as general vascular stasis, due to pathology of the temporal bone and blood vessel anomalies, affect the overburdened venous system, thus preventing absorption of the excess cerebrospinal fluid. We would like to suggest this merely as food for thought, as in a few cases where this occurred, the X-ray revealed anomalies of the lateral sinus and jugular foramen.

SUMMARY AND CONCLUSIONS.

1. X-ray examination of the mastoid process alone is not sufficient.

2. The examination should consist of a complete study of the temporal bone, including the mastoid process, the petron, the vascular structures and the jugular foramina.

3. Three per cent of skulls show an extremely small jugular foramen on one side. The lateral sinus of the same side is always smaller or may be completely absent.

4. In 89 per cent of the skulls[32] the right lateral sinus is usually larger than the left.

5. The left lateral sinus is frequently larger than the right.

6. The right lateral sinus is involved more frequently than the left, in a ratio of 3:2.

7. The lateral sinus may be completely absent. This finding has been substantiated by the X-ray preoperatively.

8. The presence of a large emissary vein may indicate the following: an absent lateral sinus; a small lateral sinus or a thrombus obstructing the lateral sinus.

9. Anomalies of the sinuses and jugular foramen occur frequently. During health the circulation is adequate and therefore there are no symptoms.

10. Symptoms appear when there is circulatory disturbance.

11. Changes in venous pressure directly influences intracranial pressure and this may be either increased or decreased.

12. Vasostastis as the result of pathology of the temporal bone, plus inadequate venous structures, will produce increased intracranial pressure and secondary meningeal symptoms.

13. We have carefully correlated the X-ray, clinical and operative findings, and we have found that the knowledge gained from the X-ray, disclosing the size of the venous structures, has helped us in the prognosis and treatment of otitic complications.

14. We may suspect a stormy course when there is a demonstrable marked difference in the size of the lateral sinus and where the larger side is involved.

15. Where the infection occurs on the smaller side, the prognosis is more favorable and resolution is more apt to occur.

16. In sinus thrombosis, papillitis is present in 10 to 25 per cent of the cases. Should a papillitis be absent then we may assume that there is adequate circulation.

17. The presence of inadequate venous circulation, plus a papillitis and choked disc, indicates increased intracranial pressure and measures to reduce it should be immediately instituted.

18. The Queckenstedt test as utilized in the diagnosis of sinus thrombosis is only of value when there is a rise of spinal pressure following jugular compression (a negative Tobey-Ayer). A positive Tobey-Ayer test must be carefully evaluated for the reasons mentioned in the text.

19. It is possible that Gradenigo's syndrome may be explained in part to venous disturbance.

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1915 Spruce Street.

PELLAGRA — ORAL AND PHARYNGEAL MANIFESTATIONS. REPORT OF A CASE.*†

Dr. Oscar Rodin, Brooklyn.

It is well established that the incidence of pellagra, food supply and consumption are closely related. In times of unemployment and general economic stress we see food deficiency diseases, especially pellagra. The United States Public Health Service reports that some 200,000 persons had this condition in 1929.[1] Although most of these cases occur in the South, pellagra is found in every State in the Union.

Of interest is the fact that this disease is also being reported among the well-to-do. One investigator reports twenty-five such cases.[2] These people so unbalanced their diets that they became defectively nourished and developed pellagra.

Two cases of pellagra which developed as a result of restricted diet after operation for gastric ulcer have been reported.[3]

Pellagra has been encountered complicating dieting, alcoholic addiction,[4] gastric lesions,[5] diseases of the intestinal tract,[6] and in patients who were put on a strict ketogenic diet.[7]

Pellagra received its name from Frappoli, an Italian, in 1771.[8] It comes from the Italian words "pella," meaning skin, and "agra," meaning rough. It has been defined by Osler[9] as a deficiency disease characterized by gastrointestinal disturbances, skin lesions and a tendency to change in the nervous system.

Goldberger[10] definitely proved it was caused by a deficiency in certain articles of food. These are:

1. Vitamin G or pellagra preventive.[11] *2.* A defective or inadequate mineral supply.[12] *3.* A physiologically defective protein (amino-acid) supply.[13] *4.* Poor food consumption.

The length of time a person can live on a defective diet without having pellagra depends upon the degree of the deficiency of the

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diet and the amount of pellagra preventive factor, vitamin G, that the individual may have previously stored.

Diets which contain milk, lean meat, fish, fresh eggs and yeast are the best anti-pellagra diets. It has been found that the addition of two to four ounces of dried skimmed milk or one pound of evaporated milk, or two ounces of wheat germ, or one and one-half pounds of cured lean pork, or one ounce of pure dried yeast to the daily food is sufficient to greatly reduce the incidence of pellagra.

In the past, too much dependence has been placed upon the presence of dermatitis or a history of dermatitis for diagnosis of this condition. Cases without typical skin lesions, the so-called "pellagra sine pellagra," are increasingly common.

The gastrointestinal tract is always involved in pellagra. The tongue, buccal mucosa, palate and pharynx show definite changes. It is for this reason that these cases very frequently come to the attention of the laryngologist first.

The mouth lesion is usually as follows: The tongue is invariably involved. During the onset of the disease it is found furred and coated, and the patient presents a foul breath. Anorexia usually accompanies this picture. The lingual epithelium is lost and the tongue becomes red and slightly swollen. Irregular fissures form on its center and upon the lateral margins. This stage has been called dissecting glossitis. The true pellagra tongue is the tongue without a coat—the beefy tongue. Aphthous ulcers may develop on the tip and margins, as a result of which pain is caused by the slightest movement.

The gums are inflamed, tender, spongy and bleed readily, as in scurvy. The outer border of the lips is dry while the inner borders of the lips and cheeks are tender, red, raw and swollen. This process extends over the buccal mucosa to the tissues of the palate and pharynx. Aphthous ulcers are common and arise as blisters, which burst and leave raw ulcerated areas. The pharyngeal lesion may spread to the esophagus, causing a sensation of rawness and pain on swallowing.

Case Report: T. G., male, age 38 years, was admitted to the service, Feb. 10, 1932, complaining of a soreness of the throat which has lasted one week. This soreness had spread to his oral cavity. His mouth felt warm and he experienced a burning sensation.

He appeared emaciated. Examination revealed numerous whitish patches upon the surface of the pharynx, hard and soft palate, the

tongue, buccal and gingival mucosa. The exudate could be readily removed, leaving a raw ulcerated surface which bled easily. There were no palpable cervical glands.

The story of the study of this case during the first thirteen days of his stay in the hospital is of interest. Upon admission, no definite diagnosis was made, but the following conditions were considered: Vincent's angina, lues, pemphigus. On the sixth day, the diagnosis of pellagra was suggested. At this time the dermatogolists suggested the diagnosis of Vincent's angina, tuberculosis ulceration or thrush.

On the eleventh day, the condition of the mucous membranes was much improved.

On the thirteenth day, the patient presented a skin lesion on the dorsum of the hands and the back of the neck. The dermatologists now made a positive diagnosis of pellagra.

On the nineteenth day, the oral and pharyngeal lesions had entirely disappeared, but the dermatitis was still present.

Ten days later (twenty-ninth day), the patient was discharged completely recovered.

During his stay he was fed a highly nutritious vitamin containing diet.

Studies included: Wassermann, sputum, chest X-rays, blood cytology and numerous smears.

Summary: A case was admitted with local manifestations in the oral cavity and pharynx. These lesions were difficult to diagnose until the true nature of the general disease, pellagra, was evident by subsequent well recognized skin lesions.

This report iillustrates that we must always bear in mind local manifestations of a systemic disease when we encounter ulcerations such as those described above. In times of depression, pellagra must be kept in mind.

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893 Eastern Parkway.

PEMPHIGUS BEGINNING IN THE LARYNX. REPORT OF A CASE.*†

Dr. Hyman Danish, Brooklyn.

A case of pemphigus beginning in the larynx is presented to emphasize that this condition may invade the larynx, the pharynx or the oral cavity weeks or months before the skin manifestations of this disease become evident. The condition is intractable and usually fatal, and when an exudative lesion is met with in the mouth or larynx, the possibility of pemphigus should be considered. Pemphigus has been reported in the larynx, involving the epiglottis and arytenoids. An unusual case of stricture of the esophagus following pemphigus at the cricopharyngeus was reported by Imperatori. In its early stage the lesion appears in the form of a small vesicle or bleb. As a result of trauma incidental to mastication, swallowing or coughing, these vesicles rupture soon after their formation. This may occur within a few hours and for this reason the typical bullous lesion of pemphigus may not be seen in the larynx or pharynx unless early and frequent observations are made. After rupture the seropurulent fluid which escapes evaporates and forms a superficial dirty gray membrane which can readily be brushed away. At times the lesions heal spontaneously in one area and are seen to develop in other areas. Cases have been recorded where the laryngeal or oral lesions have persisted for as long as three years before the skin lesions appeared. The tonsils and vocal cords appear to be uninvolved. In a majority of cases, once the cutaneous surfaces become invaded, the course is rapidly fatal.

There is nothing definitely known as to the etiological factors. Some contend that the disease is due to an infectious agent, while others believe it to be due to trophic disturbances. The disease most frequently occurs in elderly people who are debilitated.

This condition is commonly mistaken for a Vincent's infection, but in pemphigus there is no granular ulceration such as is characteristic of the former, and smears for Vincent's organisms are negative except when there is secondary invasion. Vincent's angina

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responds readily to therapy, while pemphigus resists all forms of treatment. Other conditions from which pemphigus must be differentiated are syphilis and tuberculosis, and at times leukoplakia. In each case the history and laboratory findings will serve to aid in the diagnosis. The following case is reported:

Case 96161, 1932: G. N., male, porter, age 65 years, born in England. When first seen he was complaining of slight but persistent soreness on swallowing for the past three weeks. There was nothing significant in his past history. Indirect examination of the larynx revealed an exudative lesion on the left side of the epiglottis. This was very small and resembled a vesicle. Pemphigus was immediately thought of. Other causes were to be ruled out. Smears for Vincent's infection were repeatedly negative. There were no palpable cervical glands. The blood Wassermann was negative. Blood chemistry, blood counts and smears were negative. X-ray of chest showed no evidence of tuberculosis. Five weeks later an exudate was seen on the left side of the epiglottis and tongue base. The buccal mucous membrane of the right side was also involved. Laboratory tests were repeated and were again negative. Two weeks later there were bullae on the soft palate, tonsillar pillars and uvula. These bullae ruptured, leaving a dirty gray membrane. Other bullae appeared up on the tongue, the gingival mucous membrane, the posterior pharyngeal wall and the buccal mucosa of the left side The tonsils were not involved. Swallowing was painful because of the excoriations, and the burning and smarting were intense. Examination four months after the onset of his symptoms revealed typical pemphigus lesions on the skin of the face, neck and chest. The patient was transferred to the Skin Service. Subsequently, the eruptions extended to the extremities, the back and the genitalia. He became bed-ridden, depressed and exhausted. He developed suicidal tendencies and died ten months after the appearance of his first laryngeal lesion.

7825 Fourth Avenue.

CAVERNOUS SINUS THROMBOSIS OF OTITIC ORIGIN. REPORT OF A CASE.*†

Dr. Joseph G. Gilbert, Brooklyn, N. Y.

The following is a report of a case of cavernous sinus thrombosis arising as a result of an aural infection.

E. D., a white female, age 43 years, was admitted to the hospital on May 14, 1932. She had a moderate discharge from the right ear for the preceding five months, associated with some pain in that ear for several weeks. There was no history of chills. A week prior to admission she had a severe right-sided temporal headache. Her past history was irrelevant except that she had a nasal discharge for several years.

On the day of admission the patient vomited for the first time. Examination revealed an obese white female in a confused state, generally hyper-active, and tossing about in bed. There was tenderness to pressure over the entire skull, more especially over the right mastoid region.

The upper lid of the right eye was markedly edematous; the conjunctiva was injected; the eye was proptosed; extraocular movements were limited; the pupil reacted to light and accommodation; the veins of the fundi were full, and there was slight blurring of the optic disc.

There was moderate edema of the upper lid of the left eye. There was slight proptosis and limitation of motion in this eye. The pupil reacted to light and accommodation, and the fundus was normal.

There was moderate mucopurulent exudate in both sides of the nose. Polyps were to be seen in both nasal chambers and there was a marked postnasal drip. There was no evidence of acute congestion and edema.

The right ear drum presented a small nipple in the upper part of the anterior quadrant through which foul smelling pus exuded.

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Compression upon the right jugular vein caused no increase in discharge through the drum. There was exquisite tenderness on pressure over the right mastoid area. The left ear was normal.

The reflexes were as follows: There was a Hoffman on the right side; both knee jerks were hyper-active, as well as the ankle-clonus. The Babinski and Kernig reactions were positive on the right side and the right side of the body was weaker than the left.

The temperature varied from 100.8° to 103° before operation. Lumbar puncture revealed a clear fluid under a pressure of 40 mm., mercury. During compression upon the left jugular vein, the pressure rose to 55 mm.; during compression upon the right jugular vein, the spinal fluid pressure remained at 40 mm., indicating a definite block on the right side. A smear of the spinal fluid revealed no organisms, and the cell count was normal.

Blood examination showed R.B.C. 4,400,000, and 85 per cent hemoglobin; 10,800 W.B.C. with 86 per cent polymorphonuclear cells.

The X-ray report of Dr. Rendich follows: "Both mastoids are of the pneumatic type and of moderate size. The left mastoid is well illuminated. There is a slight absence of illumination of the cells of the right mastoid. The infiltration is more pronounced in the regions of the antrum. A small area of destruction is noted in the retroantral region. The sinus film reveals a moderate involvement of the right maxillary sinus. The conclusion is right mastoiditis with destruction."

A preoperative diagnosis of cavernous sinus thrombosis arising from a right mastoiditis was made. It was believed that the infection spread through the sigmoid and then through one of the petrosal sinuses to the cavernous sinus. The possibility of this condition arising through the carotid venous plexus was considered.

Although the patient's condition was precarious and the hope for surgical cure was very slight, operative interference was decided upon because of the headache, vomiting and exquisite tenderness over the mastoid region.

At operation there was considerable bleeding from the outer cortex. Free pus was found in all the cells of the mastoid. The sinus plate was eroded and the sinus wall had a gangrenous appearance, was hard, firm and did not pit on pressure with a probe. Upon incision of the sinus wall a greenish clot with free pus was to be seen in its lumen. Free bleeding occurred from the knee of the sigmoid

sinus. The superior petrosal sinus bled freely when it was injured while trying to expose it. The incision in the sinus wall was continued down to the jugular bulb, but no bleeding was encountered from this part of the sinus. The emissary vein was bifid in character and was thrombosed.

Microscopic examination of the wall of the sigmoid sinus revealed fibroblastic tissue markedly infiltrated with small round cells. Attached to this was an organizing thrombus. In some places necrotic cellular debris was present. Study of the pus from the mastoid cavity revealed a streptococcus and a gram negative bacillus. The pus from the ear showed a staphylococcus aureus and a streptococcus.

On the day after the operation the temperature rose to 107° and the patient expired.

COMMENT.

It is unfortunate that an autopsy was not obtained. However, it was felt that we were dealing with a case of cavernous sinus thrombosis because of the history, the physical findings and the operative findings. The most probable route of the infection to the cavernous sinus was from the aural disease on the right side by direct extension from the lateral sinus through the inferior petrosal sinus. The para nasal sinuses were discarded as an etiological factor after the mastoid operation. The following facts were significant:

1. The history of a discharge from the right ear for five months accompanied by headache on the same side for several weeks. This was associated with exquisite tenderness over the mastoid process.

2. The Tobey-Ayer[1] test was positive.

3. The X-ray findings indicated destruction of bone.

4. The finding of pus in the mastoid process, an erosion of the sinus plate, a sigmoid sinus thrombosis, with free bleeding from above and none from below.

Although the X-ray study disclosed disease of both ethmoidal areas, there was no evidence of an acute process in the nose. The operative findings were too definite and too significant to be disregarded from the standpoint of etiology. The findings in this case exclude the carotid venous plexus in either the acute or chronic form as the probable pathway.

The existence in this case of an otitis of twenty weeks' duration is of considerable interest. Eagleton[2] reported a case of cavernous

sinus thrombosis arising from an ear infection in which the evidence of this condition appeared some eight weeks after the acute illness began. Turner and Reynolds[3] cited a case, previously reported by Fraser and Dickie, of cavernous sinus thrombosis which developed about eleven weeks after the acute onset of otitis media.

Of twenty-two cases of cavernous sinus thrombosis recorded by Turner and Reynolds,[4] five (22 per cent) followed middle ear disease, while twelve (54 per cent) had their inception in the para nasal sinuses.

Seven (28 per cent) of Eagleton's[5] twenty-five cases of cavernous sinus thrombosis followed middle ear infection and a similar number and percentage came from the para nasal sinuses.

Smith[6] collected one hundred and forty cases of cavernous sinus thrombosis, of which thirteen (9 per cent) arose from the para nasal sinuses and fifty-six (40 per cent) from the middle ear.

Brunner[7] has called attention to the fact that in twenty-two autopsies on cases of lateral sinus phlebitis, twelve were found to have had a cavernous sinus thrombophlebitis.

CONCLUSION.

A case of cavernous sinus thrombosis, complicating acute otitis media, is reported. This case again demonstrates that it is essential that the pathway of infection be understood if treatment is to be properly directed.

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947 Montgomery Street.

CARBUNCLE OF NOSE; OPHTHALMIC VEIN PHLEBITIS; OPERATION FOR CAVERNOUS SINUS THROMBOSIS; RECOVERY. REPORT OF A CASE.*

Dr. E. Jefferson Browder, Brooklyn.

H. M., age 23 years, white male, was admitted to Kings County Hospital, April 25, 1932, complaining of a carbuncle on right side of his nose. Five days prior to admission he noticed a small pimple on the right lower aspect of his nose, which slowly became larger in size, spreading over the right malar area and causing considerable discomfort. He was treated at home until the day of admission, when X-ray therapy was applied over the involved zone. Following the X-ray treatment the upper lid of the right eye became swollen. Two days later there was noted definite prominence of the right eyeball with marked edema of the right upper lid, and severe headache. At this time examination of the fundi showed a very definite early edema of the right optic nerve head with marked engorgement of the retinal veins, but no hemorrhages. The left pupil and the left fundus were considered to be within normal limits. The movements of the right eye were markedly limited and when attempting to move the bulbs voluntarily considerable pain was experienced. Slight rigidity of the neck could be demonstrated. Culture of pus from the carbuncle showed staphylococcus. The same organism was grown from the blood. The spinal fluid showed twenty cells per cmm., with a trace of globulin.

It was quite evident that the infection was slowly progressing, already having involved the veins of the right orbit with questionable extension as far back as the cavernous sinus. Having previously discussed the possibility of occluding the cavernous sinus with electrocoagulation, it was thought that this would be an ideal case for this procedure. Therefore, through a right cranial opening the right cavernous sinus was exposed by elevating the right temporal lobe. With the Bovie electrocoagulation unit the sinus was completely obliterated up to the orbital fissure, cranial wound was closed without drainage. The zone of the carbuncle was inspected and the

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angular vein opened, disclosing an abscessed cavity which extended into the interior aspect of the orbit.

On the day following operation there was marked edema about the entire right orbital area with extreme proptosis of the right bulb. Patient was in fair condition with the exception of marked perspiration. There was no evidence of involvement of the left eye. During the next five days the extreme degree of swelling of the right orbit remained about the same, except for the formation of three localized areas of pus which necessitated draining. The marked general perspiration continued. Gradually over a period of six weeks' time the swelling about the right orbit subsided, although the drainage from periorbital incisions continued for about two months. At no time during his course was there any evidence of involvement of the left side.

The final examination showed complete blindness of the right eye. The appreciation of heat and cold, pin prick and touch over the right side of the nose is unimpaired. The extra ocular movements are well performed.

96 Joralemon Street.

BULLET WOUND OF THE RIGHT EAR CANAL; HEMATOMA OF THE SOFT PALATE AND PTERYGO-MAXILLARY SPACE; TRACHEOTOMY AND LIGATION OF THE COMMON CAROTID ARTERY; RECOVERY; REPORT OF A CASE.*†

DR. HERMAN RUBIN, Brooklyn.

F. D., age 63 years, Italian, male, was admitted to Kings County Hospital on June 2, 1932. He was found in a cemetery beside the grave of his son, where he had attempted suicide.

The patient was in shock at the time of admission. His mouth was partly open and he was having considerable respiratory difficulty. There was considerable retraction of the suprasternal and supraclavicular regions. Active bleeding was to be seen from the right ear canal. The entrance of the bullet was found to be about 1 cm. behind the right tragus and in the anterior canal wall. The canal wall was markedly swollen, so that the middle ear could not be visualized. The wound of exit could not be found. The mouth could be opened to only about one-half inch. Pressure upon the tongue revealed a hematoma of the soft palate about the size of a small orange, which in turn was pressing upon the posterior surface of the tongue. This accounted for the respiratory difficulty. The bluish discoloration of the hematoma extended along the inside of the right cheek to a point opposite the lower canine teeth. Externally, there was a swelling which extended from in front of the ear over the parotid gland to the soft tissues over the anterior maxillary wall. The swelling extended below the jaw and occupied the anterior triangle of the neck down to about the middle of the sternomastoid muscle.

An infusion of 400 cc. of a 5 per cent glucose solution and appropriate stimulants were given. Tracheotomy under novocaine anesthesia was immediately performed. As soon as an opening was made

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in the trachea, breathing ceased but returned after one or two minutes of artificial respiration.

It was decided to ligate the common carotid artery at once because the hematoma was increasing in size and injury to a large blood vessel was suspected. Under local anesthesia, an incision was made along the anterior end of the sternomastoid, the deep cervical fascia in its upper portion was found distended with blood and bluish in color. The carotid sheath was opened and the common carotid

Fig. 1. X-ray showing numerous small fragments of bullet in temporomandibular region.

artery was ligated with two sutures of chromic catgut. A small rubber tissue drain was left in the lower end of the wound.

Progress of the Case: A Roentgenogram taken June 4, two days after the accident, showed the presence of many small metallic fragments of a bullet scattered throughout the right mandibular region near the angle of the jaw. A round mass was seen in the region of

the soft palate, which was interpreted to be the large hematoma pressing on the tongue base. The temperature rose to 103° on the fourth day after operation. The patient complained of pain in the right ear. His respirations were easy and regular and the swelling of his soft palate had diminished considerably. On the fifth day he developed signs of myocardial weakness, which responded to digitalis and other remedies.

About this time considerable purulent discharge appeared from the fistula behind the tragus (the entrance point of the bullet). A probe passed into this fistula revealed irregular bare bone of the anterior canal wall. A tract was found which extended downward, forward and inward. At the same time, a discharge was noticed coming from the middle ear. The drum membrane could be examined only with

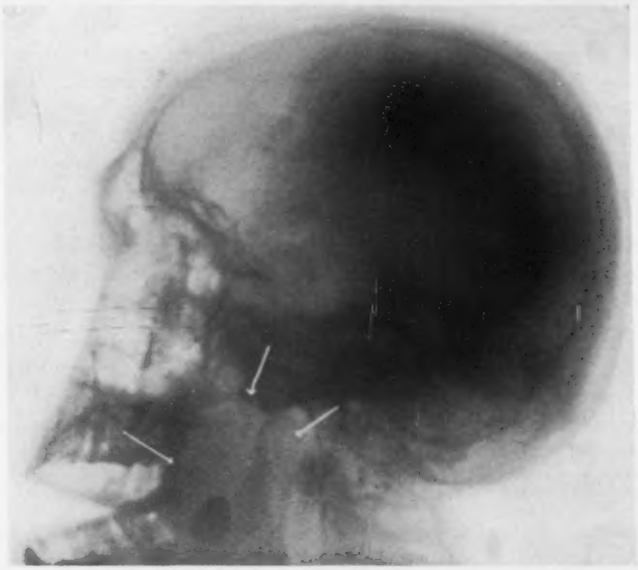

Fig. 2. Arrows indicate extensive hematoma of the soft palate.

an infant speculum because of the swelling of the canal wall. Examination showed a mucopurulent discharge coming from a small perforation of the drum anterior to the handle of the malleus at about its center.

The tracheotomy tube was removed on the tenth day. Immediately

after the ligation of the common carotid artery, the eye grounds showed considerable pallor. Later examination of the eye grounds by Dr. Hargitt revealed no vascular changes of the right eye as compared with the left. There was no pallor of either disc or either fundus. The pupils were equal, regular and responded to light. No paralysis or weakness of the opposite face, arm or leg was present.

On June 21 the right ear was still discharging profusely, and there was tenderness over the mastoid. X-ray study showed an infiltration of the cells with some absorption of the septa. The tenderness and ear discharge gradually diminished and had disappeared at the time of the patient's discharge from the hospital, July 30, fifty-eight days after admission. The hearing in this ear, several weeks later, was found to be normal for the whisper and conversation.

Comment: This patient had a bullet wound through the right ear canal, which caused a hemorrhage into the pterygomaxillary space and a hematoma of the soft palate. The latter necessitated tracheotomy on account of the respiratory difficulty which ensued. It was difficult to ascertain just what blood vessel had been injured. For that reason it was decided to ligate the common carotid artery. From an examination of the anatomy of the parts involved in this case, it is believed that the external carotid artery at its terminal portion, or the internal maxillary artery in the pterygomaxillary space, or both, were injured. Ligation of the external carotid artery might have been sufficient to control the hemorrhage into the soft palate and pterygomaxillary region.

The patient evidently had a good collateral circulation in the brain. Although there was extreme pallor of the optic nerve-head and retina, immediately after ligation, these structures gradually assumed a color similar to those of the other side. There was no opposite facial, arm or leg weakness seen.

201 Eastern Parkway.

PERITONSILLAR ABSCESS; RETROPHARYNGEAL ABSCESS; OSTEOMYELITIS OF THE BASE OF THE SKULL; EXTRADURAL ABSCESS AND DEATH. REPORT OF A CASE.*†

DR. JOSEPH S. SILVERBERG, Brooklyn.

The following case illustrates what may occur from pus trapped in the retropharyngeal space.

Case Report: A white male, age 45 years, was admitted with a history of sore throat and pain in the right side of the neck of one week's duration. The right tonsil was displaced towards the midline from a peritonsillar inflammation. This area was incised. About one dram of pus was released.

The patient appeared to be doing well until the seventh day, when there was a rise in temperature to 101°. At this time the right posterior cervical glands were palpable.

Probing of the right peritonsillar space revealed no pus. On the following day bulging was seen in the right posterior pharyngeal region but no definite fluctuation was felt. This swelling was incised. A few drops of thick pus were obtained. On the next day the right retropharyngeal space was explored with a clamp. No pus was found.

The progress appeared to be satisfactory until the seventh day. The swelling of the right posterior cervical glands had increased and the patient complained of severe pain in the throat. There was also an elevation of temperature. An external incision along the posterior border of the sternomastoid muscle was made. About one dram of pus was obtained. The tissues were found to be friable and matted together. There was excessive bleeding, so that it was found necessary to pack the wound tightly. A few days later an attempt was made to remove the packing but the wound had to be repacked

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because of excessive bleeding. The necessity for packing again occurred three days later. At this time a submaxillary incision as advised by Mosher[61] was made but no pus was found.

On the twenty-second day in the hospital, part of the packing from the posterior incision was removed. There were four additional attempts to remove the packing, but each time, because of the recurrence of bleeding, it was necessary to repack the wound. Finally, on the thirty-first day of hospitalization, the packing was entirely removed, with no bleeding. For the following ten days the patient continued to show a slight elevation of temperature with a discharge of serosanguineous fluid from the neck wounds. There was still considerable swelling of the posterior pharyngeal region on the right side.

On the forty-second day the incision at the posterior border of the sternomastoid muscle was again opened and a small amount of pus was released. Examination of the tissue submitted to the laboratory at this time disclosed necrosis and infiltration with inflammatory cells.

On the forty-fourth day, impairment of breath sounds was noted in the right chest. There was also flaccidity of the left upper and lower extremities with convulsive twitchings of the left half of the body. The patient finally expired on the forty-fifth day of hospitalization.

Autopsy Findings: Meninges: A moderate congestion of the pial and subpial vessels was noted.

On stripping the dura from the base of the skull, a few spicules of necrotic bone came away in the region of the right posterior fossa. A circular opening with necrotic edges about the size of a twenty-five-cent piece was seen. This was situated about one-half-inch to the right of the foramen magnum. The opening began along the occipital bone, extended forward to the posterior portion of the sphenoid and reached the lower portion of the temporal bone. It extended laterally to the posterior border of the parietal bone and reached as far as the petrous portion of the temporal bone. The dura covered the edges of this circular opening in the base of the skull and was adherent to it. The finger was passed into and through the wounds in the neck to the under-surface of the base of the skull. It entered an irregular cavity filled with greenish pus; this

61. Mosher, Harris P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. Trans. Amer. Acad. Ophth. and Otolaryngol., p. 34, 1929.

cavity completely surrounded the foramen magnum but did not involve its contents, as the dura limited it. Posteriorly the finger reached three-quarters of an inch to the left of the external occipital protuberance. There was also a cavity extending three-quarters of an inch to the left of the center of the spinal column into the retropharyngeal space. No opening into the pharynx was found. The attachments to the base of the skull of the recti capitis posticus major and minor on the right side were destroyed. Accumulations of disintegrated necrotic material were removed from the cavity. It was particularly noted that the dura had fully limited and demarcated the infectious process.

Summary of Histologic Data: Cervical tissue: suppurative inflammation; brain: early exudative meningitis, extradural abscess: lungs: confluent bronchopneumonia; occipital bone: osteomyelitis.

The sequence of events in this case was peritonsillar abscess, retropharyngeal abscess, osteomyelitis of the base of the skull, extradural abscess and terminal pneumonia.

This case strikingly illustrates the powerful tryptolytic action of unevacuated pus upon living structures, hard or soft.

The repeated tight packing to control the bleeding prevented ample drainage and probably favored the occurrence of the cranial and intracranial complications.

5201 14th Avenue.

LUDWIG'S ANGINA. REPORT OF A CASE.*

DR. WILLIAM J. HOCHBAUM, New York.

Although Ludwig's angina usually follows dental caries, alveolar abscess, abrasions and dental injections, it must be borne in mind that it may follow acute tonsillitis. The possibility of a tonsillar etiology is seldom considered. The following case from the jaw service of the King's County Hospital illustrates this:

Case Report: A colored male, age 24 years, was admitted with a history of having had the lower left third molar extracted eight days prior to admission. The patient talked with difficulty because he could not open his mouth.

There was a generalized swelling of the left side of the pharynx and peritonsillar area. The corresponding left side of the face was edematous. There was exquisite tenderness over the entire left cheek and neck, especially along the border of the left mandible. There was a firm discrete nonfluctuant mass at the angle of the jaw. The diagnosis on admission was osteomyelitis of the jaw with cellulitis of the neck. Within a few hours there was some difficulty in breathing. The uvula was about six times its normal size, and the soft palate of the left side was edematous. Apparently there was a left peritonsillar abscess. An incision was made through the anterior pillar. No pus was obtained but the patient felt greatly relieved. On the following day there was some difficulty in breathing, with marked edema and swelling of the tongue, which occluded the oral cavity. The floor of the mouth was elevated because of the induration in the submental region, which spread laterally and upward as far as the left maxilla. Laryngeal examination was unsatisfactory because of the edema of the surrounding soft tissues.

Two days after admission, respiratory difficulty was more marked and the patient was more septic. The local process had advanced. Cervical incision was performed for a Ludwig's angina. Although there was some apparent relief following operation, the patient became progressively weaker and a few hours later expired. Death was not due to respiratory obstruction. The autopsy findings are of interest. There was edema and swelling of the soft parts of the

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superior mediastinum. This was continuous with the edema and swelling of the tissues of the anterior part of the neck and extended almost down to the root of the pericardium. The tongue and larynx were removed in toto. There was an abscess about the size of a walnut, which appeared to be walled off and not draining, about one-half inch behind the left tonsil. This cavity contained about a half-ounce of dirty yellow pus. The pharynx, larynx and soft palate surrounding the left tonsil were swollen and edematous.

The mandible showed no evidence of osteomyelitis. The soft parts of the floor of the mouth were swollen and edematous.

Postmortem Diagnosis: Ludwig's angina, retrotonsillar abscess and terminal bronchopneumonia.

This case illustrates the importance of carefully examining the tonsillar and peritonsillar regions in infections of the mouth. It is recalled that this patient had a definite acute peritonsillar inflammation at the time of admission, which, because of the swelling of the tissues of the floor of the mouth, was disregarded in the management of the case. The peritonsillar abscess probably pointed posteriorly so that the usual anterior incision failed to reach the suppurative cavity.

1130 Park Avenue.

NUTRITIONAL AND BIOCHEMICAL PHASES OF OTOLARYNGOLOGY.*

DR. MERVIN C. MYERSON, New York.

Nutritional and biochemical considerations are not new to otolaryngology. For many years Stucky paid a great deal of attention to these phases of the specialty in Kentucky, while Shurly has done likewise in his section of the country. The many advances in our knowledge of foods and body chemistry makes it possible for the otolaryngologist to apply some of the things he has learned.

A good deal of what follows has no scientific explanation at the present time but can be stated only on a basis of clinical experience. Many substances were used in medicine for long periods of time before the scientific basis for their value was disclosed. In the consideration of this subject one must bear in mind that other things besides actual nutrition and chemical change influence tissues. Hereditary factors, psychological reactions and bacteria are only a few of these. It should also be kept in mind that the specialist should have the co-operation and assistance of the internist in this phase of his work.

The writer is eager to give credit to Jarvis and his correspondence group of otolaryngologists throughout the country, who have done so much to develop this subject in recent years. Most of what follows has been learned as a result of this group study. Only such things that have been proven by the experience of the writer will be included in this paper.

Turck[1] has called attention to the fact that the upper respiratory tract mucosa coming from the endodermic layer of the embryo which forms the primitive gut, is the most sensitive and most responsive tissue in the organism. If this is so it should be expected that the nasal mucosa would reflect definite changes in the body chemistry. It is not strange then that in many instances the otolaryngologist is able to use the appearance of the nasal mucosa as an index of his patient's well being, as an indicator of the state of the individual's nutritional and chemical balance. Stucky and Shurly, and no doubt

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others, realized a long time ago that a healthy soil nourishes a healthy plant. Applying this to their patients they soon learned to look to the food supply of the individual for the cause of frequent nasal and aural infections. Stucky fed his patients the famous Stucky soup, while Shurly used a pure vegetable mixture.

Jarvis[2, 3] has called attention to the red and pale septum syndromes. The red septum indicates a color and texture that are caused by the ingestion of too much acid ash-producing foods. This picture also follows the ingestion of too much acid medication, and is seen in conditions of fatigue, and acidosis due to disease. The red septum may be slightly red or a dark crimson red, depending upon the degree of acid saturation of the tissues. I have observed that the darker the redness the drier is the mucous membrane. It is my belief that in most, if not all, of these red septum cases dehydration probably plays an important part. We see this in severe diabetics and in individuals suffering from wasting disease. These red septum cases complain of languor, dryness of the skin, and dryness of the mucous membranes of the nose and throat. Most are underweight. They frequently complain of a pain or tight feeling behind the nasal bones or a similar feeling in the pharynx. In most cases nasal examination reveals no evidence of local sinus disease. Many have a noticeable redness of the margins of the eyelids. Investigation of the dietary reveals that the individual is eating an excess of acid ash-producing foods, such as eggs, meats, fish, bread, cereals and salty foods, and an insufficient amount of alkaline ash-producing foods, such as fruits and vegetables.

These patients are helped by changing their food intake so that they are receiving a relatively larger quantity of alkaline ash-producing foods. To hasten the return to a normal state, medications such as sodium bicarbonate and various alkalinizing preparations are of value. A beneficial result both from the standpoint of the local picture and the general physical condition will be obtained during a period of a few days to a few weeks. In most of the red septum cases three weeks or more are required in which to obtain results.

The following is the story of a pronounced red septum case:

Case 1: H. R., age 28 years, male, lawyer, complained of a tight feeling in his nose and excessive dryness of the nose and throat. Examination revealed a dark red septum, a dry skin and a redness of the eyelid margins. There was no evidence of suppurative sinus disease. A half-teaspoonful of bicarbonate of soda twice daily and

the addition of raw fruits and vegetables to the diet caused a complete change within a few weeks. The patient, who was pale and underweight, gained weight and felt better and appeared healthier. After the sixth week the bicarbonate of soda was discontinued.

One should keep in touch with his patient for excessive alkaline intake will cause symptoms just as excessive acid intake did before. In the nose the striking sign of an excessive alkaline state is the pale septum. This pallor is not that of an anemia. The pallor of an anemia is to be found in other tissues, while the pallor we are considering is localized to the nasal cavity, and is not accompanied by a similar pallor of the membranes of the oral and pharyngeal cavities. There are different degrees of paleness of the septal mucosa. To me the pale septum always indicates an excessively moist nose. These patients always have the subjective sensation of nasal obstruction even when examination reveals a fairly good airway. In extreme cases the tissues are so waterlogged that the nose is completely occluded by the increased size of the inferior turbinates. These patients sneeze, and may or may not have a copious thin mucoid discharge. Polypi are frequently found in pale septum noses and acid medication influences their shrinkage. The writer is reminded of a recent experience in which a patient with polypi in his left nasal chamber was to return for their removal two weeks after his examination. He was advised to take fifteen minims of dilute nitrohydrochloric acid three times a day in the meantime. He returned to report that his nasal obstruction had been overcome to such an extent that he decided to postpone operation. Examination revealed that the polypi had shrunken to less than one-fourth their former size. I have not seen polypi in a nose where a red septum was consistently present.

Patients with pale septa sometimes have urticaria. In many cases, also, a personal or family history of hay fever or asthma can be elicited. The so-called vasomotor rhinitis and allergic cases practically all have a pale septum. Beckman considered that hay fever patients had an alkalosis and on that basis gave them acid medication. He reported beneficial results in 40 per cent of his cases.

The treatment of these cases when they are due to poor food selection consists in changing the diet so that there is a greater acid ash-producing content. A more rapid result is obtained when dilute acid, such as nitrohydrochloric or hydrochloric is prescribed. Sodium chloride and sodium citrate produce the same effect. It is interesting to note that one can overcome the moist pale septum state or alkaline state more rapidly than the dry red septum state. The same medica-

tions which are suggested for overcoming the above syndrome are of great value in the treatment of patients with so-called vasomotor or allergic noses. Not all cases are benefited by this form of therapy as it is used at the present time. In those cases which are not benefited, the allergist and the internist will assist in solving the problem.

The early stage of a common "cold" is ushered in by an excessively moist nose in most cases. Shea has suggested that the administration of a teaspoonful of table salt will overcome the local condition and will frequently abort the oncoming infection. This has been proven to be correct, not only in the experience of the writer but also that of many of the members of the Jarvis group. The effect of the salt is felt within one to ten minutes when the moist nose becomes a dry one. The same effect is obtained by using the medications mentioned above. It would appear that the substances suggested in the treatment of the moist pale nose accomplish their result by a dehydration effect. In the case of a beginning "cold" this dehydration effect is probably sufficient to alter the local nasal condition so that infection is avoided. The subject of the red and pale septum appears to be intimatcly related to the matter of the permeability of blood vessels, and the various factors that influence increase and decrease of permeability.

Case 2: Miss S., a nurse, age 33 years, complained of nasal obstruction of four years' duration. This obstruction was associated with frequent sneezing spells and an excessively moist nose most of the time. Examination revealed that both nasal passages were completely occluded. The floor of either side of the nose was filled with the redundant lower portions of the inferior turbinates. Sodium citrate in doses of fifteen grains three times a day was given. Improvement was noticed on the fourth day. When seen at the end of a week the inferior turbinates were of normal size and the previously waterlogged nose was only slightly moist and no longer obstructed. At the end of two weeks the patient had a fairly normal appearing nose.

Carbohydrates seem to cause more rhinologic upsets than any other element of food. While poor food selection has been the cause of trouble in many cases, excessive carbohydrate ingestion or impaired tolerance of the nasal tissues for the end-products of carbohydrate metabolism have been the most frequent causes of trouble in patients presenting themselves with nasal symptoms.

There is a large group of individuals who develop nasal symptoms when they indulge too freely in sweets or starchy foods. These

individuals develop what I choose to call acute nasal disturbance due to carbohydrates; that is, their symptoms follow very closely upon the ingestion of carbohydrates, more especially the acid-forming ones, which are readily soluble, such as candy. The second group or chronic nasal disturbance due to carbohydrates are caused by an excessive intake of starchy foods or the tissues of the nose and nasopharynx seem to tolerate the products of carbohydrate metabolism poorly. It is to be noted that the same factors cause different reactions in different individuals.

The first group develop stuffy noses with or without excessive secretion of a watery consistency. This lasts for a few minutes to a few hours. If the general resistance is good this passes away; if not, this spells the beginning of a "cold."

The second group have a definite symptomatology and definite nose and throat findings. They complain of either nasal obstruction or postnasal drip. Children complain of the obstruction, while adults complain of the postnasal drip. Jarvis has noted impaired hearing in some of these cases, which is greatly improved when the diet is corrected. Examination of the nose in the second group discloses that the floor of each nasal cavity and that of the nasopharynx contain a large amount of thick, glary, colorless, or grayish, sometimes a whitish gray, mucus. This may be the only finding in some cases, especially in children. In addition there are very fine strings of mucus stretched across the nasal cavity anteriorly, from the inferior turbinate to the septum. These are very fine and are usually multiple. In the well established case of this type the posterior pharyngeal wall contains a varying amount of lymphoid tissue. My conception of this lymphoid tissue is that it is Nature's response to irritation of low grade, which is the result of the effect of excessive carbohydrate ingestion or poor local tissue tolerance. These patients complain of frequent "colds." In a large majority of cases careful examination of the nasal structures does not disclose the presence of suppurative sinus disease. X-ray study of the sinuses is usually negative.

Investigation of the diet of these patients reveals that candy is frequently eaten, or that the patient is eating too much acid-forming carbohydrates, or that he is on an ulcer diet, which requires the feeding of cereals and potatoes in relatively large quantities.

Of interest is the fact that treatment based upon the above conceptions brings definite and good results. Some of these patients,

particularly the children, have a pasty and pale appearance. In addition some have a marginitis of the eyelids. It is this group who, in addition to eating a diet too rich in carbohydrates of the wrong type, are getting an insufficient amount of vitamin-containing foods. Raw fruits and raw vegetables are given to these individuals, and in addition some acceptable vitamin preparation. The result of this therapy when combined with a reduction of the starchy foods and the prescription of a preparation which has been found to assist carbohydrate tolerance is gratifying.

Spintrate, which is a concentrate of spinach, is one of the preparations which influences the effects of carbohydrate ingestion favorably in these patients. As pointed out by Allen,[1] green vegetables contain myrtillin, a substance which has the property of increasing the tolerance for carbohydrates. A brief note on this substance is pertinent here. Allen states that innumerable vegetable substances, often in the form of powders or teas, made from green plants have been used as popular remedies for diabetes throughout the world. In 1925, Drurig was impressed with the use of blueberry leaf tea in diabetes. He suggested an investigation of this substance. As a result Wagner and Marks demonstrated that blueberry leaf tea had a definite influence upon the alimentary hyperglycemia of dogs. Myrtillin is the substance in the plant that tends to lower the blood sugar. Later, Allen and Wagner demonstrated that myrtillin occurs in all green plants. They found that this substance does not reduce the fasting blood sugar but that it reduces or suppresses the hyperglycemia which follows the administration of large quantities of dextrose to dogs and human beings.

During the winter months there is a special need for giving vitamin-containing foods to our patients. This is more applicable to those who have been operated upon or who have been ill for a long time. I have been following Shurly's[5] suggestion and have been feeding my patients a mixture of raw vegetables and orange juice three times a day. The postoperative course of our cases has been favorably influenced by this mixture. Wounds that do not granulate or heal poorly are stimulated to heal well.

The following cases illustrate excessive carbohydrate intake or poor carbohydrate tolerance and their management.

Case 3: A. S., male, age 17 years, complained of nasal obstruction, sneezed a great deal, and felt that he had a constant "cold." He has an almost constant postnasal discharge. Questioning concerning

his diet revealed that he ate pies and cakes with practically each meal. Examination revealed an increased pallor of the nasal muous membranes with excessive moisture and thick, glary mucus in the posterior nares. Excessive sweets in the form of cake was eliminated from the diet, and spintrate and dilute nitrohydrochloric acid were prescribed. Within six days he reported that he felt better and that his local nasal condition was much improved. At the end of three weeks he had lost his postnasal discharge.

Case 4: M. W., male, age 37 years, was referred by an internist for antrum irrigation because of his postnasal discharge, which was causing a beginning wheezing respiration. Examination revealed no evidence of antrum disease but there was thick grayish mucus in the posterior nares, fine mucoid strings between the septum and inferior turbinate on either side and some lymphoid tissue on the posterior pharyngeal wall. Questioning concerning his diet revealed that he ate some candy and liked his bread. Spintrate and a reduction of the carbohydrate intake cured the patient of his beginning wheeze and his postnasal discharge in less than a week.

The importance of inquiring into the diet of our patients should be emphasized. Patients who are on an ulcer diet are frequently deprived of vitamins or are receiving an excess of carbohydrates. The following cases are cited:

Case 5: J. F., age 20 years, on an ulcer diet for eight weeks. Came to the hospital with extensive ulcerations of the tongue and of the mucous membrane of the oral cavity. All laboratory studies proved negative. The ulcerations healed rapidly when he was fed with a mixture rich in vitamins.

Case 6: H. M., male, age 39 years, was on an ulcer diet which contained a liberal quantity of cereals and potatoes. He complained of an annoying postnasal drip. This was cleared up in less than a week by the administration of one teaspoonful of spintrate daily.

A case of pellagra whose only symptom was a sore throat of several days' duration was reported this evening. The importance of bearing in mind the possible local manifestations of systemic disease has already been stressed.

Summary: Some of the best understood nutritional and biochemical phases of otolaryngology have been touched upon. More work will be done in this field before the final word is spoken and this paper should therefore be regarded in the light of preliminary remarks.

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12 East 86th Street.

ETHMOIDITIS: ITS VARIOUS FORMS AND THEIR TREATMENT.*†

DR. LOUIS S. DUNN, Philadelphia.

Much has been written on the ethmoid capsule, and a variety of opinions have been expressed by different authors regarding the treatment of ethmoid disease. My personal observations of the success that may be obtained by the proper management of these conditions leads me to write this paper.

Classification: Involvement of the ethmoid may be acute or chronic, and in either case it may, in turn, be suppurative or nonsuppurative. In order to faciliate the discussion of ethmoid disease, we have followed the grouping adopted by Skillern:[1] *1.* Acute catarrhal inflammation; *2.* acute suppurative inflammation; *3.* chronic catarrhal inflammation (hyperplastic ethmoiditis); *4.* chronic inflammation with suppuration.

Acute Catarrhal Inflammation: In every case in which there is an acute inflammatory condition of the mucous membrane of the nose, as in every acute infectious coryza, no matter what the infecting organism, the ethmoid cells are more or less affected. The result is an edematous condition of the mucous membranes. The mucosa of the uncinate process, bulla and external surface of the middle turbinate become swollen, having the appearance of myxomatous degeneration with punctiform hemorrhages on various parts of the surface. The interior of the cells becomes involved in the pathologic changes, and these changes are more likely to occur in patients whose physical condition has been undermined by some acute or chronic disease process. Hays[2] believes that the repeated colds from which many children suffer are due to inflammatory conditions of the ethmoid cells, and unless proper treatment is instituted these cells become chronically diseased. In general, the symptoms of acute ethmoiditis are those of an acute cold in the head, only more aggravated. The patient complains of a stuffy feeling in the nose. There is a profuse discharge of mucous from the nose and, as a rule, a great deal of sneezing. There may be headache due to congestion.

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The acute condition in itself is rarely serious, and the prognosis is always good unless other sinuses become involved, but it is possible for the process to spread rapidly with concomitant involvement of other accessory sinuses. What is of most consequence is the possibility that repeated infections will set up chronic hypertrophy of the mucosa, which will result in chronic ethmoiditis, the formation of polypi in the nose and excessive secretion of pus.

Acute Suppurative Inflammation: Acute empyema of the ethmoid cells per se is a comparatively uncommon condition. It is usually associated with acute suppurative disease of an adjacent sinus, or may be traced to one of the infectious diseases, as influenza, diphtheria, scarlet fever or measles. In this type of ethmoiditis the mucous membrane is deep red and covered with thick purulent secretion. In contradistinction to the simple catarrhal type of disease, this form is directly due to bacterial invasion. The symptoms are those of a particularly severe cold in the head. There is absolute occlusion of the nares, particularly in the superior portion. The inferior turbinates are sympathetically congested. Headache is constant, with occasional neuralgic pains radiating to the deeper structures of the eye. Ocular symptoms are prominent; they consist in tenderness of the bulb, pain on rotation, epiphora and orbital neuralgia. There is marked anosmia as long as the nasal obstruction persists. The constitutional disturbances are similar to those occurring during the course of a severe coryza.

The diagnosis of acute forms of ethmoiditis is often difficult owing to the marked swelling which makes satisfactory examination impossible. Clinically, the differentiation of acute ethmoiditis and a severe cold in the head cannot be made. It may be said, however, that the condition is one of acute ethmoiditis when the inflammatory condition in the general nasal cavity has subsided, while the ethmoid continues to be unduly inflamed. The prognosis, like that of acute coryza, is good, but the point to be emphasized is that each attack predisposes toward another, until a chronicity develops. It is, therefore, important that a complete rhinoscopic examination be made during the interval between attacks with the view of determining the cause of the trouble and of instituting appropriate treatment before the disease becomes firmly established.

Chronic Catarrhal Inflammation: This condition, also called hyperplastic ethmoiditis, is the commonest, most troublesome and most difficult to diagnose in its incipiency of all the ethmoid affections. It is the consensus of opinion that the causative factor is nutritional

disturbance of the ethmoid capsule, rather than inflammatory changes with bacterial invasion, such as protein sensitization and gastrointestinal or endocrine disturbance. Sage,[3] McCullagh[4] and others find evidence of a relation between ethmoid affections and endocrine imbalance. The tissue change in hyperplastic ethmoiditis is usually associated with systemic conditions which cannot be explained by nasal stenosis alone. From clinical observation, Sage concludes that in addition to hay fever, asthma and urticaria, nasal hydrorrhea and hyperplastic ethmoiditis and some forms of hyperplastic rhinitis are but varied manifestations of the reaction of the organism to protein, known as anaphylaxis. This writer finds that the symptoms which are common and nearly constant in the nasal conditions above mentioned are nearly identical with those of protein poisoning. The writer of this paper holds that polypoidal ethmoiditis or so-called hyperplastic ethmoiditis is a direct result of protein sensitization or allergy. Soft polypi as well as general polyposis are caused by sneezing, which in turn is caused by protein poisoning. A common form of allergy, which the writer found to cause the condition, is a sensitization to bacterial proteins. A common offender was found to be protein from staphlococcus albus.

Pathologically hyperplastic ethmoiditis is characterized by anaplasia of the mucous membrane of the nasal cavity, especially in the region of the middle turbinates and ethmoid bulla. The tissue change consists of mucoid degeneration of the mucosa, with infiltration of connective tissue, which contributes to the ultimate formation of polypoid tissue. The mucous glands are primarily hypertrophied and not infrequently show enormous cystic dilatation of their acini. The blood vessels are surrounded by leukocytes and soon begin to atrophy. The process extends to the bone, the periosteum becoming hypertrophied and showing fibrous degeneration along the bone. These pathologic changes are transmitted directly to the bone and subjacent tissues.

The symptoms of which the patient complains are almost continuous head cold, as in acute coryza or acute ethmoiditis. The hypertrophic form of ethmoiditis is often a part of a diffuse hypertrophic process involving the lower turbinate bones as well. Associated with this type of ethmoiditis one often finds granular hyperplastic pharyngitis, particularly behind the tonsillar pillars, as well as hypertrophy of the tonsils and Eustachian catarrh. Bronchitis of varying degrees is also a frequent accompaniment of this form of ethmoid disease, and asthma has long been recognized as a concomitant. The impor-

tance of this relationship is well shown by the numerous cures reported after removal of the ethmoid structures (Brown[5]).

If eye symptoms are associated with hyperplastic ethmoiditis they are usually of mechanical origin. The subjective symptoms consist of scotoma, neuralgic pains in the globe, ciliary neuralgia and photophobia. In severe cases there may be vasomotor disturbances, such as hyperemia of the conjunctiva and edema of the eyelids.

Chronic Suppurative Inflammation (Empyema): This type of ethmoiditis may occur as an open empyema or as a closed latent process. It is the result of an acute suppurative process, because of involvement of the bony framework, when the drainage of the ethmoid cells has been seriously impaired, or it may result from an acute infection taking place in the presence of hypertrophic ethmoiditis; the defective drainage keeps up the process as a chronic empyema. Chronic purulent ethmoiditis is invariably due to bacterial infection, the pathologic changes being similar to those of empyema in other sinuses, *i. e.*, thickening similar to those of the mucosa with marked fibrous tissue formation, and it is usually associated with disease of other sinuses. Round-cell infiltration is prominent, with gradual proliferation of the epithelium, which in severe cases is often absent in spots, being replaced by granulation tissue. There is general metaplasia where the secretion comes in contact with the mucosa. The glands are primarily atrophied, due to obliteration of blood supply. In this form of ethmoiditis the apposition of bone predominates, while in the hyperplastic type the resorptive changes are more active.

The symptoms of chronic suppurative ethmoiditis are exceedingly variable, depending not only upon the virulence of the infection and the extent of the process, but upon the general condition of the patient as well. There is often very little subjective discomfort. Frequently the patient complains of pharyngeal or laryngeal discomfort rather than of trouble referable to the nasal sinuses. In the uncomplicated form where there is free drainage there is often no history of headache, while in a closed empyema the pain is often very severe. The typical region for the localization of pain in ethmoid disease is over the root of the nose and directly over the vertex, occasionally radiating down into the mastoid process (Skillern). Deep-seated pain in the eyes or tension on the bulb is not present unless there is pressure from the accumulating pus in the closed cavity. The exudate is distinctly purulent and invariably exhibits a tendency to dry and form crusts. The quantity naturally

depends upon the extent and severity of the inflammation. There may be periods of profuse discharge lasting for days, then suddenly there will be a remission, which in turn is followed by an acute exacerbation.

Chronic Hyperplastic Inflammation with Suppuration: There is considerable difference of opinion in regard to adopting this classification. Most observers, among them Sluder, contend that the suppuration precedes, and does not follow hypertrophies. Skillern says that if this is true he has frequently overlooked the purulent stage, as in most of his cases it has been absent. Although authorities may not agree as to which of these conditions is the primary one, we know that they may exist in combination and may give rise to various symptoms associated with the pathology of the ethmoid cells.

In addition to the forms of ethmoiditis above described, Shambaugh[6] calls attention to a chronic atrophic form, which occurs as a rule in connection with a general atrophic process throughout the nose. This atrophic process is usually bilateral and apparently begins in the mucous membrane of the ethmoid; it is characterized in the ethmoid, as elsewhere in the nose, by atrophy and shrinkage of the bony framework. When atrophy is seen to be developing, especially where it is unilateral, as on the side where a deflected septum has produced a roomy nares, suspicion should be directed to the ethmoid labyrinth.

Complications: A consideration of the various sequelae of chronic suppurative ethmoiditis makes emphatic the necessity for early and accurate diagnosis and prompt and appropriate treatment in every case in which accessory sinus disease is suspected. Among the complications which may be encountered are external rupture with fistula formation, rupture into the bulbar cavity, inflammation of the lacrimal duct, cerebral complications, such as cavernous sinus thrombosis, brain abscess, meningitis, otitis media and focal infections in various parts of the body, particularly the various types of arthritis, which may occur at any time during the existence of sinus infection.

External rupture with fistula formation is the most frequent complication. This is what one would expect in view of the anatomical conformation of these parts in which the orbital structures are separated from the ethmoid labyrinth by only the thinnest possible plate of bone (lamina papyracea). The point of predilection for perforation to occur is in the region of the ethmolacrimal suture. The frequency of abscess and fistula formation in comparison with the infrequency of orbital affection is in a large measure due to the

orbital periosteum of the lamina papyracea, which offers considerable resistance to the pressure exerted by the secretion.

Acute rupture in the orbit with outward dislocation of the bulb, and swelling and infiltration of the eyelids, accompanied by intense pain, high fever and prostration, has been reported as a sequel of suppurative ethmoiditis in a number of cases. Rupture in the orbit may occur as a chronic process, coming on gradually and having none of the stormy symptoms found in acute rupture. The eye is simply dislocated little by little.

Disturbances of vision from infections of the posterior ethmoid cells are almost as frequent as those resulting from sphenoid involvement. Schaeffer[7] has made an elaborate study of the visual pathway in relation to the paranasal sinuses and describes various types of posterior ethmoid cells. He states that not infrequently the posterior group of ethmoid cells is relatively large and encroaches over and under the usual site of the optic foramen. This results in the optic foramen elongating into a true osseous canal, located in a sense within the ethmoid cell in question. In this type of case the canicular segment of the optic nerve is markedly increased in length; often the bone of the canal is defective and of paper-like delicacy, and thus it is easy to see why ethmoidal disease may produce such marked visual disturbances. Experience has shown that the longer the diseased condition continues the less is the likelihood of improvement in vision after operation.

A study of the lymphatic drainage of the accessory sinuses, such as that made by Andre[8] and Mullin,[9] makes it evident that infection from the ethmoid, as well as from the other accessory nasal sinuses, can easily find entrance into the general lymph channels and circulation, thus explaining the origin of the various complications and sequelae of chronic suppurative ethmoiditis coming under the head of focal infection.

Diagnosis:[10] In view of the many and possibly serious sequelae of ethmoid disease the greatest care should be exercised that these conditions be not overlooked because of incomplete examination. Often the diagnosis depends, as McNaught points out, on the associated symptoms or systemic reactions, rather than upon the microscopic changes in the nose. In making the examination one should first examine the middle turbinate. The objective signs are hypertrophy and anemia of this structure, of the area directly anterior to it, and of the middle meatus, with possible polypoid changes in the anterior end of the lower border of the outer surfaces of the middle turbinate.

Advanced cases show masses of polypi in the middle meatus. The uncinate process and middle turbinate should be forced apart, thereby permitting one to inspect the bulla and surrounding tissues. Posterior rhinoscopy should be employed to determine the condition of the cells of the superior nasal passage. If pus is observed to issue from the middle nasal fossa, the nose should be thoroughly cleansed with saline solution and the source of the secretion located. If there is any doubt as to its origin, the maxillary antrum should be punctured. Vogel[11] and others believe that this should be done before resorting to a roentgenographic examination, in as much as the exploratory puncture is easily accomplished, and in case the antrum is infected the procedure is of therapeutic as well as of diagnostic value. If the maxillary sinus is not involved, one should then proceed to sound and catheterize the frontal sinus, washing it out. If the returning fluid is negative a tentative diagnosis of ethmoidal suppuration can be made. In order to find the exact source of the secretion, it may be necessary to resect a portion of the middle turbinate, and this serves the purpose of drainage as well. After this wound has healed it may then be possible to observe purulent secretion exuding from the middle nasal passage. If this is seen together with the appearance of crusts in this locality, which may conceal foci of pus, the diagnosis is substantiated.

Until quite recently ethmoiditis has been considered to be essentially a disease of adult life, but it is now recognized that ethmoid disease occurs frequently in children. It has also been noted that in children otitis media and enlarged cervical glands are complications often encountered.

Transillumination as an aid to diagnosis in accessory nasal sinus disease has had its advocates, but little reference is made to it in the more recent literature. Edward Leander Pratt[12] finds it an invaluable indicator of what sinus is involved. He reports a case in which a diagnosis of ethmoiditis was made and treatment was directed solely to this region. In the opinion of the writer, transillumination is of little value in diagnosis. Had transillumination been employed and the darkness of the antrums investigated, treatment of the latter would probably have avoided the sacrifice of both middle turbinates and the ethmoid labyrinths. The common error, this writer thinks, of directing treatment to the ethmoid cells, often to the extent of sacrificing them in toto while allowing an antrum full of pus to remain neglected, is in large part traceable to failure regularly to employ or properly to interpret transillumination. The criticism that

transillumination is unreliable is based on the fallacious premise that occasionally an antrum will illuminate darkly and on puncture will be found to contain no secretion. Skillern, on the other hand, has abandoned the use of transillumination altogether in the examination of the ethmoid cells as he finds it thoroughly unreliable in ethmoid suppuration. This he attributes to the fact that it is impossible to place the light in such a position as to send rays through the bulla and surrounding structures. This is particularly true if there is present an old ostitis.

While there is still a great difference of opinion as to the value of Roentgenograms in the diagnosis of ethmoid suppuration, they are generally believed to be of greater value than transillumination. Lemere[13] states that a slight density greater than is normal has decided diagnostic value, but in children a dense shadow rarely shows. Pomeranz, in a recent article, claims that Roentgenographic examination of the sinus in the hands of a skilled Roentgenologist is an invaluable aid in the interpretation of a diseased condition and thinks that greater co-operation between the Roentgenologist and practitioner would prove of great assistance in the logical and successful treatment of a case. He insists, however, that a thorough physical examination is the first essential, since it is astonishing how many extraneous conditions simulate true sinus disease and, furthermore, intranasal conditions, such as deviated septa, enlarged turbinates, while they are often the cause of sinus disease, will present a typical sinus picture when no such pathology is present. Pfahler[14] and Skillern have had moderately satisfactory results as regards the posterior and sphenoidal cells by placing the plate under the chin and the light on the vertex, but they advise that substantiation and resubstantiation should be made. Amedee Granger,[15] after an extensive study, claims to have located landmarks which are a great aid in diagnosis of ethmoid conditions Roentgenographically, the most important being a curved line which bounds the sphenoid, called the optic groove; it curves downward on each side toward the optic foramen and the anterior clinoid process. By the use of this technique, the author believes it is possible by a careful study of a set of Roentgenograms to make earlier and more positive diagnosis of diseases of the sphenoids and ethmoids than have heretofore been possible. Summing up the present status of the X-rays as a means of diagnosis in ethmoiditis, we may say that during the past decade occasional disappointments have been experienced and it is the consensus of opinion that it is wise not to operate on X-ray findings alone, but to corroborate them by other exposures, and to confirm

the diagnosis as far as possible by clinical manifestations.

Treatment: In every case of ethmoiditis treatment should be directed toward the elimination of the infection as quickly as possible. The nasal mucosa should be thoroughly shrunk with cocain or adrenalin solution. At times cotton soaked in 20 per cent argyrol may prove very effective, allowing it to remain in the nose fifteen minutes. One must, however, be on the lookout for arygria, which may follow the above treatment. The author, however, believes that shrinking and irrigation suffices, without the application of any colloidal silver or other antiseptic. If the patient suffers excessive pain, analgesics may be administered, to which may be added minute doses of belladonna or atropin, to lessen the discharge. Operation is seldom necessary in the acute stage. In the chronic stage few patients get well without operation, though some of the earlier cases will respond to thorough cleansing of the nose. I believe the administration of vaccines is useless. After conservative methods to secure a free nasal cavity, by attention to the septum, the removal of polypi, the amputation or fracture of the middle turbinate, such methods as shrinkage, suction and the application of medicinal remedies to reduce inflammation should be given a trial before more radical intervention is attempted.

I shall not go into the surgery of the ethmoid at this time, save to say that when surgery is indicated, the external operation is advocated by Ferris Smith, of Grand Rapids, Mich., as the operation of choice. This, together with the fact that neglected ethmoiditis often leads to serious ocular disturbances, intracranial lesions and focal infections, serves to emphasize the importance of early and accurate diagnosis, and the institution of treatment based on a careful study of the requirements of the individual case.

CONCLUSIONS.

1. The diagnosis of ethmoiditis is not always easy as the symptoms resemble so closely those of a common cold.

2. Acute ethmoiditis, if neglected, may develop into a chronic condition very resistant to treatment. It is, therefore, important that every case of coryza should receive prompt and efficient treatment.

3. In cases of visual disturbance of doubtful origin it is always wise to make a careful examination of the ethmoid cells, since un-

suspected or latent ethmoid disease has often been found to be responsible for ocular conditions.

4. In the diagnosis of suppurative ethmoiditis transillumination and the X-rays may be of assistance, but the chief reliance should be placed on clinical manifestations.

5. As the results of radical operation in chronic suppurative ethmoiditis are often disappointing, every effort should be made to diagnose and appropriately treat ethmoid disease in its early stages.

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AMERICAN OTOLOGICAL SOCIETY.

Sixty-Sixth Annual Meeting, May 8 and 9, 1933.

MONDAY MORNING SESSION, MAY 8, 1933.

The Sixty-Sixth Annual Meeting of the American Otological Society, Inc., convened at Hotel Raleigh, Washington, D. C., at 9 a. m., Dr. George L. Tobey, Jr., President of the Society, presiding.

Presidential Address. Dr. George L. Tobey, Jr.

Dr. Tobey called attention to the fundamental pioneer work in otology done by many men, such as St. John Roosa, Clarence Blake, Buck, J. Orne Greene, Alexander Randall, Gruening, Whiting, McKernan, Jack, Dench and many others. Among the other achievements of the American Otological Society has been the foundation of the Research Committee on Progressive Deafness, and the compilation and publication of a complete bibliography on otosclerosis, a study and presentation of the ossification centres of the internal ear, with the demonstration of the blood supply of the labyrinth. This Society has seen the invention of the audiometer, the standardization of tuning forks, the true evaluation of the caloric and rotation tests and, to a very large extent, the researches made by McNally and others, together with the clinical work of Lewis Fisher. "The treatment of traumatic palsy promises to have been solved by the monumental work of Dr. Charles Ballance and Dr. Arthur Duel."

He called attention also to the studies on the cellular elements of the petrous pyramid and to the study of the dynamics of the cerebrospinal fluid which has demonstrated the value of the manometer test as a diagnostic procedure in lateral sinus thrombosis. The genetics of otosclerosis have been studied, and Weaver and Bray in their experiments have demonstrated a microphonic effect detected by currents of the auditory nerve. Other achievements have been effected and other researches are now being carried on. One of the most important things to be investigated is the problem of meningitis and other intracranial pyogenic diseases of otolaryngological origin. Dr. Tobey suggested that during the course of the meeting, the Society appoint a committee of leading men from each of the definite sections of the country, who may be ready or willing to organize a subcommittee in their respective centres, including one or two otologists, one or two competent neurologists, a neurologic surgeon if available, and a neuropathologist who is interested or may become interested in this subject. He suggested that a comprehensive questionnaire be prepared and submitted, the net result of this work to be submitted for discussion at each annual meeting of the American Otological Society. "This plan need in no way encroach upon the prerogatives of the individuals who are now carrying on investigations but would unquestionably be a means of saving many lives since it is the tendency at the present time to feel that something must be done, although the why and the wherefore of what to do may not be known."

Report of the Committee on Progressive Deafness. Dr. J. Gordon Wilson.

Three broad lines of research were laid down by the Bureau, which were as follows: (a) anatomical investigation of the petrous temporal; (b) genetic investigation of progressive deafness; (c) experimental and metabolic investigation, including endocrine, bearing on the production of progressive deafness.

This program was interpreted as allowing of flexibility, therefore a line of purely physiological investigation has been added; namely, the physiological investigation of conduction phenomena along the pathway of the VIIIth nerve. At present no funds are available to aid this latter research, but it is hoped that through the influence of the Bureau such support will be forthcoming.

Genetic Investigation: This has been carried on by Dr. Davenport, and the recent results published in the *Archives.*

Anatomical Investigation: Valuable contributions have been made under the auspices of the Bureau: These include work by Bast and Anson on the development of the human temporal bone; sections were studied, exact models prepared; comparisons were made with various mammalian fetuses, resulting in a very complete study, now partly published.

Anatomy of the petrous bones has been studied with regard to the cartilage islands, both in the human fetus and after birth, and special attention paid to their formation and postfetal changes. At present particular attention is being paid by both investigators to the Fissula ante fenestrum, especially in: (a) that it is a synchrondrosis to relieve strains, a theory much considered in Europe; and (b) its association with otosclerotic areas which arise near, or in connection with it. Working from different standpoints, Dr. Bast and Dr. Anson have been able to correlate and check their findings. Papers are being prepared and Dr. Bast has forwarded four models to our museum. Dr. Bast has also prepared an article on "Resorption of Cartilage of the Canal Portion of the Otic Capsule and Its Relation to the Growth of the Semicircular Canals." This has added to our knowledge of these primitive cavities.

Anson has made studies of the so-called cartilage islands, and their replacement by true bone. Their formation and disappearance in infancy and childhood are discussed. He has also studied the cartilaginous otic capsule in various vertebrates, especially in the stapedial region.

The structure, form and location of otosclerotic patches in adult human ears were studied from microscopic sections and by means of a model prepared by sections.

Dr. Anson has also interested several graduate students in otological research and with them has studied the comparative embryology and development of the endolymphatic duct with models, and also the comparative embryology and development of the utricular valve.

II. Experimental Work: Work on conditioned dogs has been carried on in the Department of Psychology at Illinois, and in the Department of Physiology at McGill. Dr. Culler, of Illinois, has tested the hearing of conditioned animals, following plugging of the round window. He found that opening the bulla had no effect on hearing, but that a plug placed in the fossa of the round window impairs hearing, which is restored when the plug is withdrawn. He also found that such a plug reduces the electrical pulses over the auditory nerve. This shows that electrical pulses are related to, if not responsible for hearing.

He found that firm pressure on the oval window may increase the electrical pick up from the nerve, but this is not constant.

Studies on hearing acuity in conditioned animals, as influenced by the X-ray, are still going on. So far, findings indicated an increase, due to X-ray, and this holds for some time.

Experiments on decorticated animals were difficult, owing to high mortality.

One animal was kept alive for some time and rapidly conditioned to an electric bell. This shows that something corresponding to hearing was still present.

Dr. E. P. Fowler has carried on audiometric investigations at the Medical Center, and made routine examinations of temporal bones obtained at autopsy, also those of experimental animals fed on a low calcium diet. By the ultraviolet microscope he has studied fibres of the petrous bone, and their relations to cartilage islands and otosclerotic foci. With the aid of a revolving knife he has been able to cut sections of undecalcified bone. He has also laid the foundations of an otological museum.

Dr. Marvin Jones has established laboratories for pathological studies at the Lying-in and Post-Graduate Hospitals. He has made a large collection of

infant and fetal bones and has begun the microscopic study of these. He has obtained funds for this work from persons he has been able to interest.

Report from Prof. Tait's Laboratory at McGill University.

During the past year work on the hearing of animals has been carried on by Dr. Dworkin, Dr. Sutherland and Mr. Ross.

Dr. Dworkin's Report: We have almost proved that in dogs the auditory region of the cortex is confined to the "Munk" area. Wider cortical expanse previously described appears to include areas for body tremor. (2) The cat has been used by a new method of conditioning in these experiments. Conditioned animals give the only valid test of the cortical registration of sounds. In these tests vibrating platforms have been constructed that operate in a soundless manner.

Dr. Sutherland has overcome many difficulties in conditioning experiments. He has endeavored by these means to obtain a reliable audiogram for the dog. He has improved the generation of pure high-pitched notes, and has utilized the sensitive flame, as a measure of intensity.

Mr. Ross reports on the finer apparatus for detection of electrical currents set up by the activity of the VIIIth nerve receptors. He shows that in fishes otolithic receptors serve audition. He details his operative technique for ablation of saccula or cochlea in cats, preparatory to conditioning these animals. Experienced men who work on conditioned responses feel that the real results are obtained from the unanesthetized, normal, moving subject. Reliable audiograms for each animal should be made before and after operative interference. No conditioned test on animals has any value without carefully prepared audiograms.

Dr. McNally and Dr. Tait have worked exclusively on the equilibratory centres of the labyrinth, and later will work on the auditory receptors. They feel that the former provide important leads for work on the auditory receptors.

Report from Clarke School for the Deaf. Dr. Nielson.

Studies on the auditory apparatus of deaf children have been made with a view to determine the character, location and extent of the defect. Studies also have been made on the immediate family of such children.

Two important studies have been charted. One represents the study of the descendants of one individual who settled in America in 1630, together with a less detailed study of families related by marriage to the main family. Deafness seems to be on the increase in the descendants of this family, partly because so many individuals, both deaf and hearing, have married into families in which there is a long history of deafness, have made consanguineous marriages, and partly because of some environment factor, not yet discovered.

Another chart records deafness since 1700, in certain isolated families settled in New England. Deafness reached its peak in this section in the middle of the nineteenth century and is gradually disappearing, due to scattering of the families, and marriages with different strains. However, there is, in the Clarke School, a direct descendant of one family, who had known deaf individuals in 1700, and another pupil was descended through another line. In each case, one parent was from unrelated stock.

A study of auditory acuity for eight tones was made of seventy-five pupils, and these groups were found to fall into four groups.

Group I is composed of six pupils having the largest amount of residual hearing. All hear loud speech, at least with one ear. With one exception all respond to the eight frequencies used. The hearing curve is higher at the two ends. The response for the two ears at 512 d.v. show a loss of from 40 to 70 decibels. The response to similar tones by bone conduction was near normal for some, reduced for others, but the character of the curve was the same, with a dip in the middle. The vestibular response varied from normal to slight.

Group II. Seventeen pupils with small amount of residual hearing by air conduction. Many can hear speech, or get sense of its rhythm by amplifying devices. Thirty-four ears tested showed response to highest tones in 25 per cent. The curve has a descending character. There is loss of 40 to 95 decibels, at 512 d.v. there is 65 to 95 decibels loss. B.C. shows marked dropping off for high tones. Threshold for low tones much lower than in Group I. Vestibular response was negative in seven, normal in three, diminished in twenty-four.

Group III. Forty-one pupils having practically no usable residual hearing. Curve markedly descending showed marked high tone loss. Vestibular responses varied from none in ten to normal in sixteen individuals.

Group IV. Is composed of eleven pupils having the most profound type of deafness. Four gave no response to any sound on the 2-A audiometer. The remaining seven gave responses to only two tones at maximal intensity. Threshold for sounds by bone conduction was similar to that obtained by air conduction.

Otosclerosis Complicated by Other Lesions. A Study of Roentgenograms, Audiograms, Laboratory and Clinical Findings. Dr. Edmund Prince Fowler.

A study of 126 cases of otosclerosis collected over a period of six years is analyzed, all carefully studied with the aid of the latest developments in otology, including audiograms, Roentgenograms and differential and chemical blood examinations. With few exceptions, these cases had been diagnosed as otosclerotic by other otologists, but no doubt on autopsy some would show no otosclerosis, and without doubt many cases omitted from the list will on autopsy show otosclerosis.

Thirty-nine cases of otosclerosis were studied by microscopic sections in complete series and twelve in individual slides. Patients were divided into two main groups, those without complicating nerve deafness (81), and those with complicating nerve deafness (45). In the whole series only 25 per cent showed no clear cut clinical evidence of past inflammation in the middle ear.

Each of the two main groups were divided into those: 1. Without evidence of other complicating lesions. 2. With evidence of past masked or unrecognized inflammatory lesions in the middle ear. 3. With definite history of past suppuration in the middle ear.

A fourth group with present suppuration was omitted so as not to confuse the picture.

Every patient suffered bilateral deafness, and in most instances one ear was affected before the other. There was no totally deaf ear. Past masked inflammation constituted fully 50 per cent of the complications found in both nerve and no nerve deafness with otosclerosis.

A history of progressive deafness appeared in 50 to 70 per cent.

History of colds, exanthemata and so forth was very high throughout.

Tinnitus was present in a large percentage of ears (82 to 88 per cent).

The author feels that except in the cases showing steady losses that the variations in hearing were due more to the variations in the pathology complicating the otosclerotic ankylosis than to any change in the bony ankylosis itself, and that any treatment resulting in improvement was due to improved pathology in the middle ear and nasal spaces. He believes, however, this is an approach to the therapeutic management of these cases which should not be despised.

As controls, the author gave a table showing the degree of involvement in the nasal sinuses in thirty individuals with normal hearing. In these both the

degree and percentage of involvement were low as compared with the otosclerotic cases.

The most universal evidence of past inflammation in the nasal sinuses in the otosclerotic cases, and the comparative freedom from inflammatory signs in the controls, still further strengthens the argument for inflammatory middle ear lesions as etiological factors in the deafness accompanying otosclerosis and, in fact, as a factor in the etiology of otosclerosis itself.

Roentgenograms of the ear bones show little if anything of significance, and in like manner the blood chemistry and Wassermann showed nothing of positive interest except that the otosclerotic process was present in spite of normal blood chemistry. This does not mean that at the start of the otosclerotic process some change may not have been present.

Why does otosclerosis not appear more frequently if otitis media is prevalent and a probable factor in the etiology of otosclerosis? The answer is that it does appear more often but is not diagnosed. Routine autopsies give an incidence for otosclerosis of more than one in twenty in all individuals, though few of these show bony ankylosis. This suggests that in routine autopsies in patients with deafness, the incidence of otosclerosis would be much higher. Otosclerosis pathologically is not a rarity; otosclerosis clinically is a comparative rarity only because of failure of diagnosis. Photomicrographs were shown to demonstrate the lesions showing evidence of inflammation in cases of otosclerosis. Statements made were derived from a study of five cases of otosclerosis from the author's collection in the Department of Pathology at Columbia, fourteen cases of otosclerosis obtained abroad, and thirty-four cases of otosclerosis from the Johns Hopkins Hospital.

For controls he used his entire collection of some 160 bones and a series of twenty consecutive cases studied by his son, Dr. E. P. Fowler, Jr., in the Johns Hopkins Hospital, through the courtesy of the research staff.

Summary: The clinical evidence indicates that in these cases of otosclerosis there was definite evidence of the almost universal prevalence of post-inflammatory lesions in the head spaces, particularly in the nose and middle ear, and that there were no blood abnormalities of any kind which could be linked up with the otosclerotic process. The Roentgenograms of the sinuses strongly strengthened the clinical evidence. Syphilis may also be excluded because the Wassermann reaction was positive in but a single instance. The pathological evidence substantiates the clinical observations.

Conclusions: "Progressive deafness" is not synonymous with otosclerosis because other lesions often cause progressive deafness.

Otosclerosis is not synonymous with progressive deafness because otosclerosis frequently causes no ankylosis of the stapes footplate and no deafness. All the deafness accompanying otosclerosis may not be due to bony ankylosis because there are usually accompanying complications which are capable of causing deafness.

Often all the deafness accompanying otosclerosis in certain cases may be due and has been found to be due to a nervous lesion, and have no obstructive background whatsoever. Examiners should keep in mind the fact that otosclerosis may and usually does occur coincidently with other lesions of the ear. The detection of other lesions does not preclude a subsequent diagnosis of otosclerosis. The only approach to treatment of otosclerosis which promises prevention or arrest of the lesions is treatment to combat the tendency to recurrent or chronic inflammatory processes in the nose and ear spaces, particularly the nasal sinuses, because it is herein that the foci of infection tend to linger, ready to resume again and again an active state, and so threaten or continue the inflammations, suppurations, adhesions, thrombosis and ankylosis in the middle ear.

Especially is the otologist cautioned to search for otosclerosis even though his immediate diagnosis is definitely a residual inflammatory lesion of the ear, because otosclerosis is usually accompanied by evidence of such lesions in the past.

Treatment of the complications of otosclerosis is not only indicated as the only management which may improve the deafness but as the only management which at present holds out any hope of preventing or arresting the otosclerotic process.

DISCUSSION.

DR. THOMAS J. HARRIS: This paper is very broad and touches many sides of this very important subject. There is no time in this discussion to touch on all the points mentioned, so I will confine myself to discussing the more important details. I think that one of the strong points in summing up is where he calls attention to the relation of otosclerosis to middle ear disease. That is a thing we are all familiar with. We immediately ask the question whether it is possible to make a differential diagnosis between the two conditions, or get the relationship of one to the other in an etiological way. I am concerned with the questions as to whether one of our English confreres is wrong when he says it is not possible to make a diagnosis of otosclerosis except postmortem.

I think that a number of points that Dr. Fowler makes are open to challenge. He makes an arbitrary distinction in his 120 cases between nerve lesions and non-nerve lesions. He makes his premises without proofs, but these points do not bear on the larger questions he has brought up; namely, the question of middle ear disease. I think he makes an original point when he shows the relation to previous disease in the nose and nasal sinuses. I differ from him in some of his methods of diagnosis. I think to make the assertion that *all* cases of otosclerosis must be bilateral would require confirmation that can only be obtained at autopsy. Clinically, I have seen cases in the beginning stages that only occurred in one ear. Dr. Fowler has voiced his condemnation of the Rinne test, and also stated his views in study of the low tones. There is considerable discussion on this matter, but he has gone further than most of us in this and his substitution of the ratio between air and bone conduction. However much we value the audiometer, I think we can confidently assert that some of his statements about bone conduction are open to question.

In regard to tinnitus, although tinnitus is an outstanding and constant symptom in my experience, I question if we can say that it is pathognomonic of otosclerosis, in comparison with middle ear disease. Dr. Fowler says we have failed to diagnose otosclerosis in many cases, and I agree with that.

DR. STACY R. GUILD: When one thinks in terms of organic lesions, otosclerosis is but one form of the several distinct pathological conditions which have been grouped together clinically as chronic progressive deafness. The only reason which has ever been seriously advanced for this grouping is that on inability to make differential diagnosis in a large number of cases. While there is general agreement of opinion as to otosclerosis in typical cases, the agreement is almost as general that when there is either a history of or the signs of any form of chronic otitis media it is impossible with the present methods of clinical examination to say that an otosclerotic process is not also present. In microscopic sections the differential diagnosis is perfectly easy and it may be that correlation of a sufficient number of complete clinical records with the histopathology of the same cases will enable someone to establish a truly differential point clinically for otosclerosis. But a new method of testing for the presence of bony ankylosis of the footplate of the stapes appears to be needed, one which will distinguish an osseous from a fibrous tissue interference to ossicular movements.

As to Dr. Fowler's principal thesis, that inflammatory processes in the middle ear play an important role in initiating the otosclerotic process in the otic capsule, it seems to me from the very nature of the facts he has presented to be impossible to either prove or disprove the idea so far as concerns the site of predilection anterior to the oval window.

Might I ask Dr. Fowler how, on this basis, he would account for an otosclerotic area near the fundus of the internal auditory meatus, or near the

carotid canal, or along the nonampullated ends of the vertical canals? The nature of the fibrous tissue in and near otosclerotic areas cannot well be regarded as evidence either for or against Dr. Fowler's theory, for its presence is easily and fully accounted for by the fact that the region is under the influence of the inflammatory process in the bone itself—otosclerosis being an osteitis in its essential features, whatever its etiology may be.

DR. EMIL AMBERG: In regard to the combination of otosclerosis with otitis media, Politzer's textbook contains an important reference. Dr. Fowler divides his groups into two: (a) without nerve deafness; (b) with nerve deafness. We could also classify cases as those with progressive labyrinthine deafness, and those without progressive labyrinthine deafness, which would give a further point of distinction. In regard to the calcium content, Dr. Fowler said there was no diminution of the calcium content. I should like to call attention to the admirable work by Bauer and Stein, which treats the constitutional pathology of ear disease. They quote Leicher that there are two different kinds of calcium. One is the calciumion. In pregnancy there is a lowered calcium blood content. There is a definite relation between the calcium content in pregnancy and otosclerosis, it would seem. In pregnancy the otosclerotic condition is aggravated. Another phase is the overaction of parathyroid function, which diminishes the calcium content. As to the question of bilateral involvement, Bezold, I think, said that 88 per cent of cases were bilateral. Bauer and Stein feel that the effects in the ear are produced by effects in the general system. Heredity plays a great part.

DR. LOUIS GUGGENHEIM: In my experience also, middle ear pathology has been found in conjunction with otosclerosis but, I believe, as a mere coincidence. I would like Dr. Fowler to explain why we find so many typical cases of otosclerosis where the tympanic cavity is free from any involvement, since he feels that middle ear pathology may be the cause of this condition. I would also like Dr. Fowler to explain how a middle ear involvement could bring about a bilaterally symmetrical otosclerotic process. Dr. Guild has mentioned otosclerotic foci far away from the tympanic cavity which could not be related to the tympanic process. We all look upon Dr. Guild as an authority, and I was interested in his statement that an active inflammatory process is present in bone when otosclerosis exists. None of the specimens I have examined have shown evidence of this inflammatory process. There are many cells present, including mesenchyme cells, fibroblasts, osteoblasts, histiocytes and osteoclasts, but I have never found round cells, leukocytic infiltration or any other sign of inflammation as I understand it.

DR. NORVAL H. PIERCE: The diagnosis of uncomplicated fixation of the stapes can be made during life. With a normal drumhead, patulous tube, increased bone conduction, loss of the low tone limit to above 36 d.v., decreased air conduction and a history of heredity, the diagnosis is complete. One should not try to confuse this picture. Stapes ankylosis due to otosclerosis is sometimes combined with plastic processes in the middle ear. Such cases require the nicest discrimination in diagnosis. The diagnosis of plastic stapes fixation in an individual with an otosclerotic history presents still greater difficulties. Cases of otosclerosis in which the foci are situated elsewhere in the labyrinthine capsule than about the footplate—that is, without stapes ankylosis—present the greatest diagnostic difficulties. Nevertheless, these types should not befog the fact that we are able to readily diagnose uncomplicated otosclerotic fixation of the stapes, and these are by far the most numerous of this class.

In all histologic study of the pathology of the ear we must remember that otosclerosis is not a disease of the periosteal or endosteal layers of the capsule but primarily of the endochondrial layer.

DR. EDMUND P. FOWLER, closing: My object was to put down in black and white the facts as I found them in these 128 cases of otosclerosis, and to point out what appeared to be some of the outstanding findings.

Dr. Harris asks, "Can I make a differential diagnosis?" I would say, "Probably as well as anyone else because of the very careful and complete examina-

tion. I think Dr. Harris misunderstood me when he says that I claimed all cases of otosclerosis must be bilateral. Findings at autopsy alone prove this is not so, but, nevertheless, the symptom of bilateral progressive deafness is of aid in the diagnosis of otosclerosis and I have therefore mentioned it. I did not say the Rinne was of no value. I said it was of limited value compared to accurate audiometric tests by air conduction and by bone conduction, using either the audiometer or properly calibrated tuning forks.

I lay a great deal of stress on bone conduction because, although like air conduction, it may be inaccurate, nevertheless, it enables us to attack the problem from another angle and without bone conduction data over a spread of frequencies it is impossible, in my opinion, to arrive at a diagnosis anywhere near as accurate as when the tests by bone conduction are made.

Had I not used the audiometer at all in the tests, but merely the calibrated tuning forks, the audiograms I have shown in the different types of otosclerosis would have been just the same. There seems to be an idea that there would be a difference between the hearing tests by tuning forks and the audiometer, but there are none if both these instruments are properly calibrated and properly used.

Of course, tinnitus is not pathognomonic of otosclerosis, any more than cough is pathognomonic of inflammation of the upper respiratory tract, but if constant, it, like the positive family histories, progressive deafness, bilateral, and so forth, aids in arriving at a diagnosis of otosclerosis.

I agree with Dr. Guild that it is often impossible to tell whether the middle ear involvement found in microscopic sections was very old or more recent. We know that otosclerosis may begin years before any deafness is noted and that it may persist throughout the life of the patient without there being any deafness.

Dr. Guild asked: "If sclerosis depends upon middle ear inflammation, how do I account for its being present in various sites in the petrous bone?" Well, in the first place, I have not said that otosclerosis is necessarily due to middle ear disease in whole or in part. I did say that it could not be ignored as a possible cause of otosclerosis. Even deep in the petrous tip we may have inflammation with apparently no connection with the middle ear and yet believe this to be the site from which it regularly originates.

Dr. Amberg reminds us that Politzer believed otosclerosis was caused by catarrhal otitis media. I am not stating that it is caused by one kind of otitis media or another. I am simply putting down different types of otitis media that I have found in 128 cases of otosclerosis and called attention to the practically universal occurrence of otitis found as proven clinically and pathologically.

As to the two kinds of calcium, I believe there are now claimed to be several kinds of calcium in the blood. I have no report to make from this angle as yet.

I attempted estimations of metabolic weight and so forth but found tremendous variations and nothing which seemed to lead us anywhere, at least at the time they were made.

Dr. Guggenheim asked how I can explain the findings of otosclerosis without the findings of any inflammation. Well, in the first place, I think it is seldom that no signs of inflammation are present; and second, that signs of old inflammations are apt to fade out with the years and become increasingly difficult to detect. When I have used the term "inflammation" I have not necessarily meant suppuration or even exudate inflammation. We must remember that there are various types of productive inflammations which are apt to leave nothing but a little scarring in the tissue attacked.

(To be continued in a subsequent issue.)